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VOLUME XXXI NUMBER 6

DISEASES

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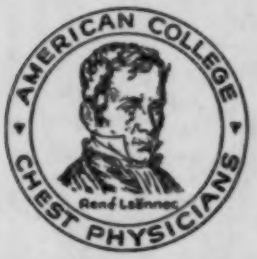

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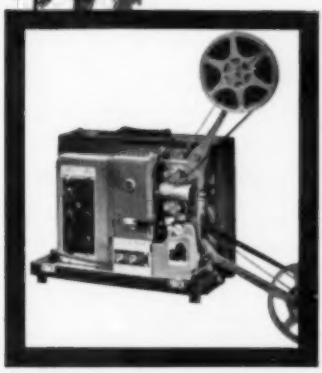

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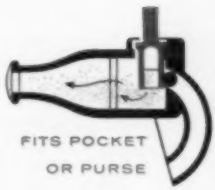

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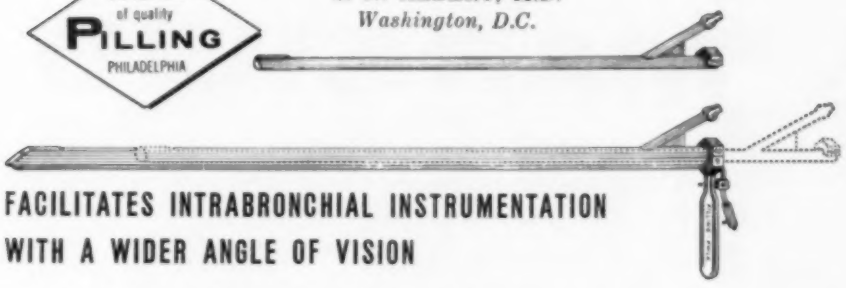

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Q. How does Marsilid act?

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Q. What are the indications for Marsilid?

A. Mild depression in ambulatory, non-psychotic patients; psychoses associated with severe depression or regression; stimulation of appetite and weight gain in debilitated patients; chronic debilitating disorders; stimulation of wound healing in draining sinuses (both tuberculous and non-tuberculous); adjunctive therapy in rheumatoid arthritis when associated with depressed

*Eudaemonia is a feeling of well-being or happiness; in Aristotle's use, felicity resulting from life of activity in accordance with reason.

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a psychic energizer

(the opposite of a tranquilizer)

psychomotor activity (Marsilid stimulates physical and mental activity, appetite and weight gain without objective joint changes).

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Q. What are the potential side effects of Marsilid?

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Q. When is Marsilid contraindicated?

A. Marsilid is contraindicated in overactive, overstimulated or agitated patients. Marsilid therapy should be discontinued two days before the use of ether anesthesia. It should not be given together with cocaine or meperidine. In patients with impaired kidney function, Marsilid should be used cautiously to prevent accumulation. Marsilid is not recommended in epileptic patients.

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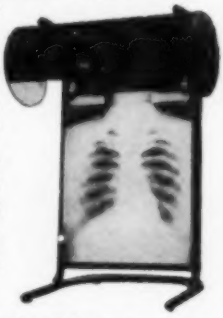

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Cohen, R. V.; Molthan, L., and Zarafonetis, C. J. D.: Clinical Studies of Various Forms of PAS (with special reference to plasma concentrations), *Diseases of the Chest* *30*:418-428 (Oct.) 1956.

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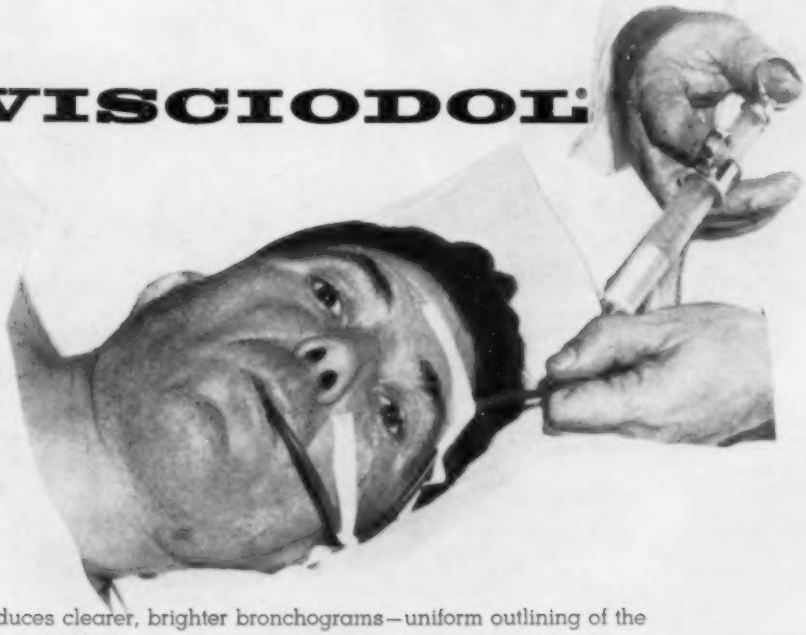

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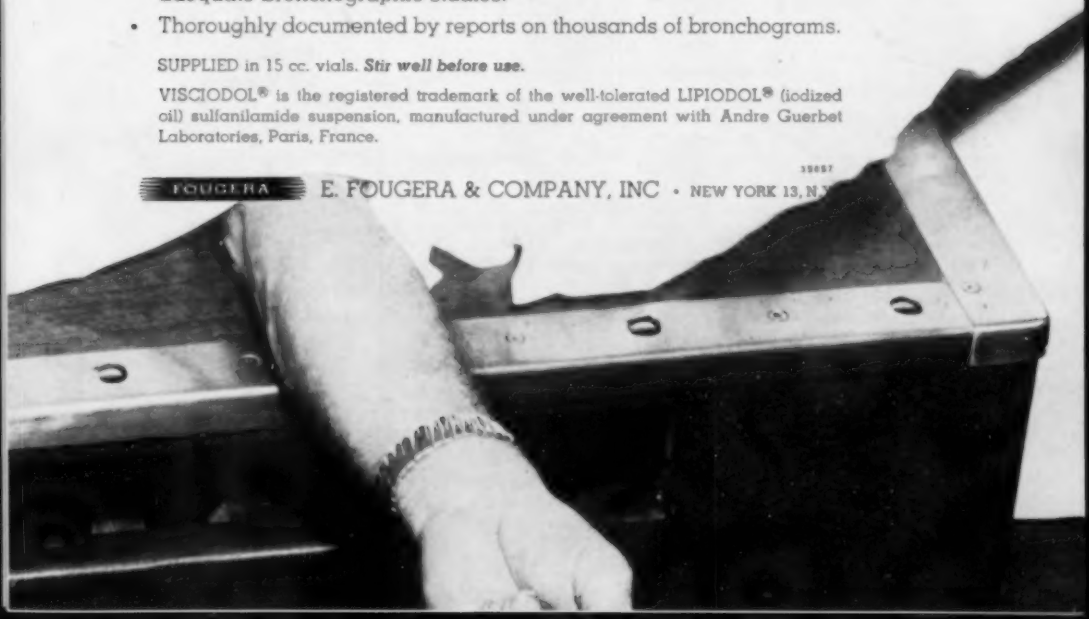

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Some of the historical aspects of the surgery of pulmonary tuberculosis have been drawn from John Alexander's book published in 1925 and 1937 and from the uncompleted manuscript for the third book on which he was working at the time of his death.

A short chapter on the surgical management of tuberculosis in the mentally ill has been included since this is an important subject and little discussed.

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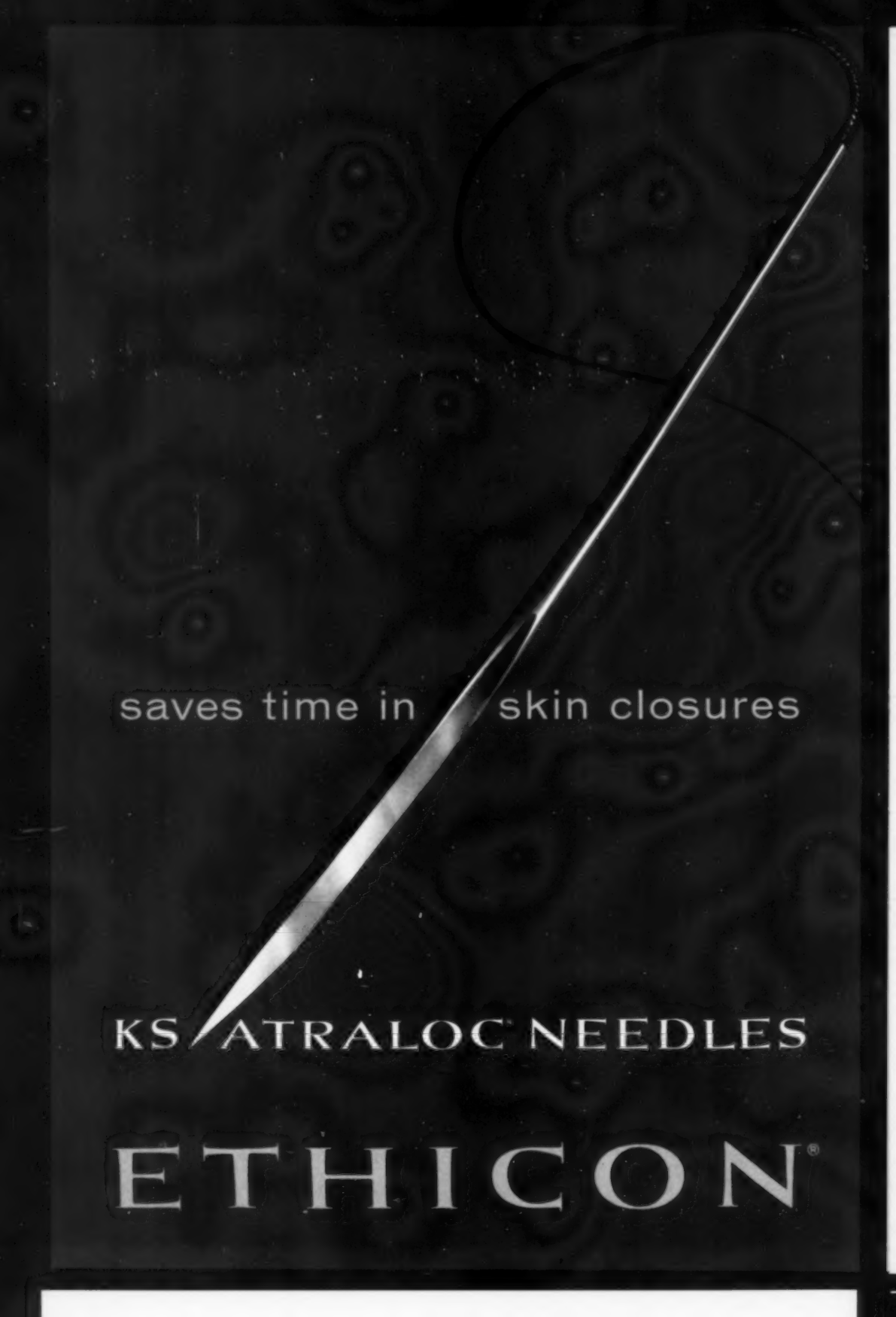
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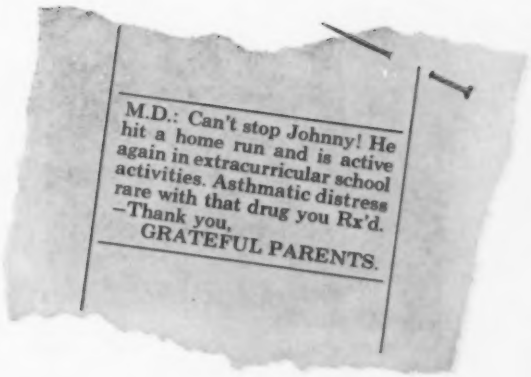
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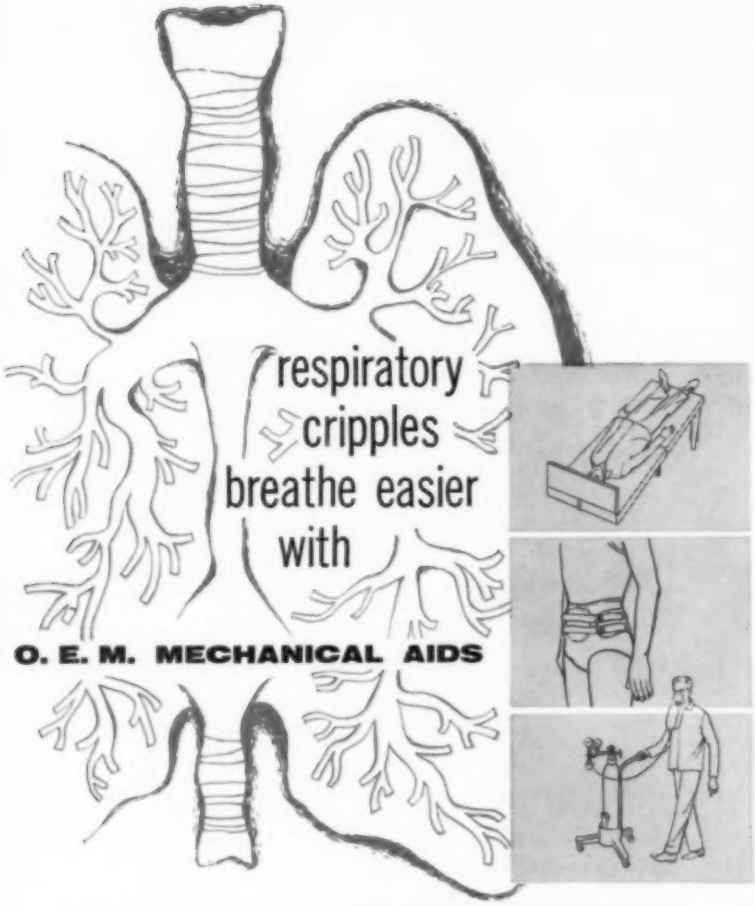

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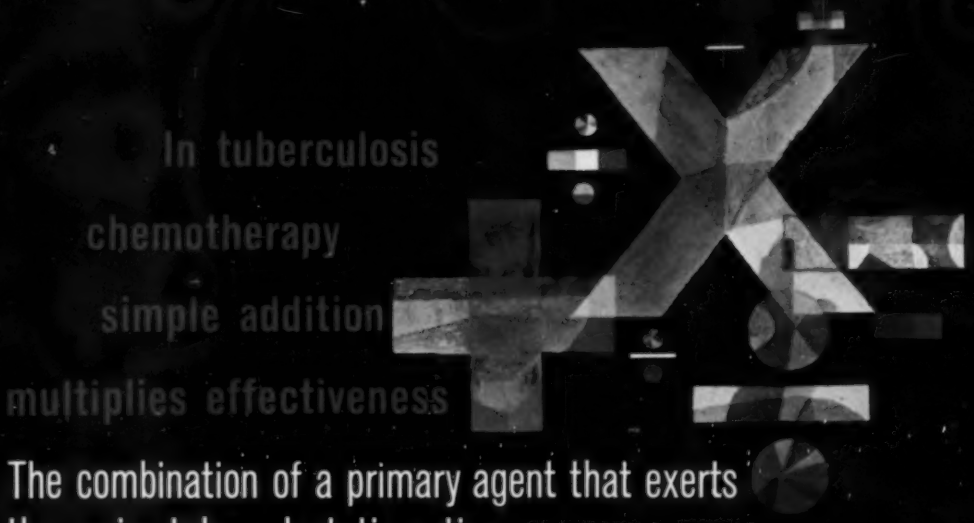

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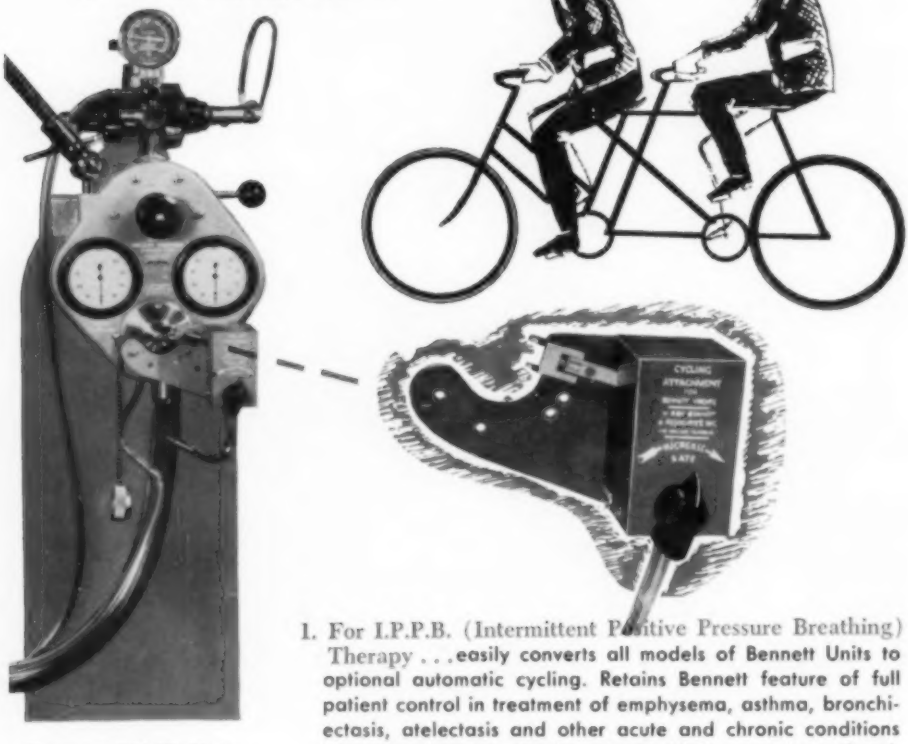

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DISEASES of the CHEST

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Clinical Aspects of Congenital Anomalies of the Trachea and Bronchi

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Congenital malformations of the trachea, bronchi and lungs may produce bronchopulmonary problems of unusual and challenging clinical interest. In the first hours, days or weeks of life, severe, acute respiratory emergencies occur as the result of congenital deformities. Some are correctable if promptly recognized, others are incompatible with life, while probably the majority are symptomless, appearing as incidental findings on routine medical, surgical or post-mortem examination. A resume of this problem is presented because of the increasing frequency with which it is being encountered and to emphasize the newer diagnostic and therapeutic aspects, both bronchoscopic and surgical, of the many corrective procedures.

Classification

A simplified, practical classification of lower respiratory tract malformations serves as a basis for this discussion.[1] It has been developed for clinical purposes and omits anomalies of monsters and malformations incompatible with life. The modified classification is as follows:

I. Anomalies of the Trachea

- A. Constrictions and Enlargements
  1. cartilage deformity
  2. anomalous vascular compression
  3. webs.
- B. Evaginations and Outgrowths
  1. tracheoceles, diverticuli, cysts
  2. fistulae
  3. tracheal lung
- C. Abnormal bifurcation or deviation

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II. Anomalies of Bronchi and Lungs
- A. Agenesis and atresia
- B. Constrictions and enlargements
  1. Webs
  2. Compression from cardiovascular anomalies
- C. Evaginations or outgrowths.
  1. Bronchoceles, diverticuli
  2. Cysts and emphysematous lobes
  3. Fistulae
- D. Subnumerary bronchi, lobes, fissures
- E. Supernumerary bronchi, lobes, fissures
- F. Anomalous bronchial and lung tissue attached to respiratory system.
- G. Anomalous bronchial and lung tissue attached to tissue other than respiratory system.
- H. Other gross anomalies
  1. Situs Inversus.

I. Anomalies of the Trachea

The most common anomalies of the trachea are the stenoses or webs which involve elements of the tracheal wall. Fibrous strictures appear as webs (Fig. 1) or as fusiform segments of stenosis with deformity

FIGURE 1: Congenital tracheal web as seen through the bronchoscope.

(Fig. 2). Absence of one or more of the tracheal cartilages results in varying degrees of dyspnea with severe inspiratory stridor but a clear cry. Lateral x-ray films of the neck and chest may give the tentative diagnosis, while bronchoscopic inspection of the dyspneic newborn infant will confirm the diagnosis. Tracheotomy may be necessary to alleviate the symptoms. Heavy cartilaginous overgrowth or other deformity of the cricoid cartilage or first tracheal ring is the most typical site of anomalous cartilage enlargement.

Stenosis of the trachea may accompany congenital esophageal atresia and tracheoesophageal fistula. In these cases the area of constriction is found at the level of the fistula between the trachea and the lower esophageal segment.[2]

Of recent interest is the frequency in which stenosis associated with cardiovascular anomalies is recognized. In this important group of cases, the heart or an anomalous great vessel constricts the trachea or a bronchus during development. Many anomalous vessels may contribute to the obstruction to cause a constricting vascular ring around the trachea. A double aortic arch is the simplest of these. In others a patent ductus becomes a part of the ring. Pressure from an abnormally originating right subclavian artery may compress the trachea to cause obstruction. These conditions have long been recognized;[3] they are frequently revealed by bronchoscopy and bronchography, during surgical exploration or at autopsy. Of greatest importance, however, from a therapeutic standpoint, is the finding of tracheal or bronchial cartilage absence or deformity at the site of vascular constriction. Removal of the obstructing vessel, or ligation and severance of a portion of the vascular ring relieves the pressure on the airway, but abnormal narrowing persists. Tracheotomy and temporary advance of a polyethylene tube through the narrow trachea or bronchus serves to give necessary support to the lumen until the wall becomes rigid or firm enough to maintain itself as an airway. (Fig. 3)

Several types of tracheal evaginations are recognized in this study; tracheoceles are demonstrated as the cause of severe respiratory and esophageal obstruction both by means of roentgen study and endoscopy. Resection is the only therapeutic procedure to be considered, giving complete relief of symptoms. It would appear that such cysts are pinched-off aberrant bronchial or tracheal buds in view of the character of their mucosal lining. They are of early embryonic origin.

The common form of tracheal evaginations are the fistulae associated with congenital esophageal atresia. A congenital fistula without atresia is relatively rare but falls into this category. Other tracheal evaginations consist of accessory bronchi with their pulmonary lobes or segments originating directly from the trachea; an accessory right upper lobe bronchus is most frequent.[4] This may appear as a complete duplication of the right upper lobe or as a tracheal origin of only the apical segment and its bronchus. Of five such cases in this series, the anomaly was an incidental finding in four while in the fifth, a congenital lung cyst in the anomalous lobe led to bronchoscopic and bronchographic studies that revealed the nature of the anomaly.

Abnormal bifurcation of the trachea has been observed in two cases. In one, a four month old infant, the bronchoscopic examination showed the right main bronchus to originate from the medial wall of the left bronchus opposite the orifice of the left upper lobe. This observation was confirmed on surgical exploration, done because of tracheal compression. An enlarged thymus was found compressing the trachea. In addition, there was an anomalous subclavian artery lying anterior to the thymus. In the second case of abnormal bifurcation of the trachea, an infant with multiple congenital anomalies, both a right and left upper lobe originated directly from the trachea with three lobes in each lung.

II. Anomalies of the Bronchi and Lungs

Anomalies of the bronchi and lungs consist of various forms of agenesis, duplication, constriction and evaginations. Complete agenesis of one lung is fairly common and has been observed in five cases;[5] in two, the absence of the right lung and in one, the left, was found shortly after birth when bronchoscopic examinations were requested because of dyspnea, absence of breath sounds, density on chest x-ray film and marked shift of the heart and mediastinum. An atelectasis of an entire lung, possibly due to a mucus plug had been suspected. In the fourth case, the absence of the left lung was found on examination of a seven year old boy suspected of having aspirated foreign body. In the fifth case, the absence of the left lung was found at post-mortem examination associated with an absence of the left half of the diaphragm, the abdominal contents filling the left hemithorax. The autopsy also confirmed the diagnosis in two of the first three cases, while the third is still living; multiple congenital anomalies

FIGURE 2: Deformed, flattened tracheal and bronchial cartilages causing fatal obstruction.

were associated with the bronchopulmonary anomaly in three that died. Early embryonic disturbances were evidently the cause.

Constrictions or enlargements of the bronchi follow the pattern of similar anomalies of the trachea. Congenital bronchial webs, bronchomalacia and compression or constriction due to cardiovascular anomalies may be responsible for severe respiratory distress. The bronchial deformity which arises during embryonic development through a constricting anomalous vessel was found to be of particular importance. Actual deformity or permanent absence of cartilage is noted in these cases. This is of clinical significance as it explains why surgical removal of the obstructing vessel may reduce the degree of obstruction, but does not necessarily eliminate it entirely. Dilatation and temporary support of the bronchus by the placement of a polyethylene tube beyond the site of the obstruction should be reiterated. (Fig. 3)

Bronchial evaginations or outgrowths consist primarily of fistulae, bronchogenic cysts, diverticuli, bronchoceles, and the numerous congenital cystic malformations of the lungs. The latter constitute a large variety of lesions which present many symptoms and unusual gross and microscopic findings. They may be associated with bronchial as well as vascular anomalies. Large solitary cysts, for example, may be found in an accessory lobe or segment, attached to the bronchial system by an anomalous bronchus. Rapidly expanding cysts may be responsible for acute dyspnea in infants necessitating emergency thoracotomy and lobectomy because the expanding cyst compresses the remaining thoracic contents. The thoracotomy should be preceded by bronchoscopic inspection in order to rule out the possibility of a congenital bronchial stenosis or web.

An extremely interesting clinical entity, somewhat similar in its action to the congenital tension cysts has recently been recognized.[6] The condition consists of an emphysema of one lobe which gradually increases the size of the lobe to cause compression of the remaining pulmonary tissue. Generally observed in the newborn period, the clinical picture is one of dyspnea and cyanosis with x-ray films suggesting a severe obstructive emphysema of the affected lobe. (Fig. 4) The absence of bronchoscopic evidence of bronchial obstruction, either by extra-bronchial compression or intra-bronchial web formation is an indication for a thoracotomy and removal of the emphysematous lobe. Histologic study of such surgically-removed lobes has demonstrated an alveolar fibrosis which may represent an important etiologic factor. The condition cannot be clinically differentiated from obstructive lobar emphysema due to vascular compression of a bronchus or to bronchial cartilage abnormality other than by bronchoscopic inspection or surgical exploration.

Supernumerary bronchi, lobes and fissures are fairly commonly encountered abnormalities, although description of these anomalies obtained from a review of the literature, as well as an analysis of our own records, show that aberrant lobation seldom coincides with bronchial variations. Pulmonary hypoplasia, in the form of subnumerary lobes, is more frequently associated with other serious developmental defects than were other anomalies herein described.

Supernumerary lobes, bronchi and fissures were most frequently observed as accessory lower lobes. Supernumerary fissures without additional lobes are common surgical observations and of little clinical significance, the superior and medial basal segments being most frequently established as separate lobes. An accurate statistical evaluation of the frequency of these anomalies is difficult to obtain, since the finding is merely incidental and not apparent prior to surgery or post-mortem examination.

Anomalous lung tissue attached to respiratory organs is relatively rare. An anomalous lobe with no bronchial communication was recently observed, receiving its aeration through direct alveolar continuity with adjacent pulmonary tissue. Because of increasing dyspnea, the emphysema was recognized and the lobe resected.

Anomalous bronchial and lung tissue attached to other than respiratory organs is occasionally observed. An example of this type of anomaly, reported elsewhere,[7] consisted of a bronchus and lung tissue originating from the esophagus. The fistulous bronchial tract was observed esophagoscopically, and the diagnosis confirmed on thoracotomy and lobectomy. Embryologically, such a case could indicate a strange and rare anomaly. It could be explained on the assumption that the primitive foregut had retained or acquired multiple potentialities of forming tracheobronchial evaginations. Most likely, the aberrant primordium had arisen, in very early embryonic life, close to the normal laryngotracheal groove but,

FIGURE 3

FIGURE 4

Figure 3: Polyethylene tube inserted through tracheotomy into right lower lobe bronchus of six weeks old infant maintaining patency of bronchus following resection of compressing vascular ring. Absence of cartilage at site of vascular compression necessitated temporary bronchial support.—*Figure 4:* Emphysematous left upper lobe causing extreme cardiac and mediastinal shift in an infant two weeks of age. The resected lobe showed severe emphysema and an alveolar fibrosis.

secondarily, had become separated from it by the growth in length of the esophagus.

Discussion

The diagnosis of congenital malformations of the lower respiratory tract during life is made with the help of various services and the team work of pediatrician, roentgenologist, bronchologist and thoracic surgeon. The history of rubella during the first trimester is important. From the physical findings one may differentiate a laryngeal anomaly from other obstructions to respiration by the character of the cry and by direct laryngoscopy. In evaluating a pulmonary lesion suspected of being a congenital anomaly, physical findings of the chest must be correlated with the interpretation of the x-ray film. Specifically, an obstructive emphysema of one lung with compression atelectasis of the opposite lung, requires such correlation to be differentiated from an obstructive atelectasis of one lung with compensatory emphysema of the opposite lung. An erroneous interpretation will be obtained if one relies on a single chest film, the report stressing the atelectasis of a relatively uninvolved side rather than the much more significant obstructive emphysema of the affected side. Fluoroscopic study, x-ray films in inspiration and expiration, a lateral view with the arms down and back to reveal the larynx, a barium swallow, bronchography, planography, and angiocardiography will add significant information in special instances. Bronchoscopy without anesthesia is now an accepted diagnostic procedure in unusual respiratory conditions in infants. Limiting the time of the procedure, the use of small instruments and the administration of oxygen through the scope during the examination increase the margin of safety. Finally, exploratory thoracotomy in infants, now a feasible, well established procedure, increases the scope of diagnosis and adds therapeutic possibilities which were unavailable until recent years.

As in most studies of congenital anomalies, the review of anomalies of the lower respiratory tract has shown frequent associations with anomalies of other organs and systems, as well as a frequent multiplicity of anomalies within the respiratory tract itself.[8] The formative genesis of many of these anomalies can be explained on the basis of an understanding of the normal embryology of the lungs. The causative factors, in most instances, remain obscure. It is emphasized that truly congenital anomalies arise early in embryonic life, most likely before the end of the second month of intra-uterine development. Abnormal conditions arising during the later fetal life are pathologic rather than teratogenic in nature.

SUMMARY

Congenital anomalies of the lower respiratory tract are described and classified and the clinical aspects are then discussed. Anomalies of the trachea include constrictions and enlargements, evaginations and outgrowths and abnormal bifurcation or deviation. Anomalies of the bronchi and lungs which are described include agenesis, constrictions, evaginations, subnumerary and supernumerary bronchi, lobes and fissures and anoma-

lous bronchial and lung tissue attached to the respiratory or other systems. The clinical significance of the anomalies is found to be greatest in the newborn period when the acute respiratory symptoms they may cause require prompt diagnostic studies, including bronchoscopy and exploratory thoracotomy. Particular stress is placed on the clinical picture caused by varying degrees of bronchial obstruction due to cardiac and vascular malformations. The discussion emphasizes the importance of teamwork between pediatrician, roentgenologist, bronchologist and thoracic surgeon in the management of the interesting problem presented, and the fact that when one gross anomaly is found, there are frequently other serious malformations of other systems that should also be recognized.

RESUMEN

Se describen y clasifican las anomalías congénitas de las vías respiratorias bajas, así como se discuten sus aspectos clínicos.

Las anomalías de la tráquea incluyen constricciones y ensanchamientos, evaginaciones e hipertrofias, y bifurcación anormal o desviación.

Las anomalías de los bronquios y de los pulmones que se describenson: agenesia, constricciones, evaginaciones, bronquios subnumerarios y supernumerarios, así como lóbulos y fisuras, y tejido bronquial y pulmonar anómalos adheridos a los aparatos respiratorio u otros. La significación clínica de las anomalías, se encuentra que es mayor en el período del recien-nacido cuando los síntomas respiratorios agudos que ellas causan pueden requerir un diagnóstico pronto mediante broncoscopía y toracotomía exploradora. Se destaca principalmente el cuadro clínico que causan grados diversos de obstrucción bronquial debida a malformaciones cardiacas y vasculares.

La discusión enfatiza la importancia del trabajo en equipo incluyendo el pediatra, radiólogo, el broncólogo, y el cirujano de tórax para hacer frente a los interesantes problemas que se presentan y el hecho de que cuando se presenta una anomalía grande hay frecuentemente otras serias malformaciones en otros aparatos o sistemas, debe tenerse presente.

RESUME

Les auteurs décrivent les anomalies congénitales de l'arbre respiratoire inférieur, les classent, et en discutent les aspects cliniques. Les anomalies de la trachée comprennent les sténoses et les dilatations, les évaginations, les excroissances et les bifurcations anormales ou déviations. Les anomalies des bronches et des poumons qui sont décrites comprennent l'agénésie, les étranglements, les évaginations, les bronches absentes et surnuméraires; les lobes et scissures, les anomalies des tissus bronchiques et pulmonaires liées au système respiratoire et aux autres systèmes. Les auteurs trouvent que la conséquence clinique des anomalies est plus grande dans la période qui suit immédiatement la naissance, lorsque les symptômes aigus respiratoires qu'elles peuvent causer nécessitent des études diagnostiques rapides, comprenant la bronchoscopie et la thoracotomie exploratrice. Ils insistent particulièrement sur le tableau

clinique que réalisent les divers degrés d'obstruction bronchique causée par les malformations cardiaques et vasculaires. Ils mettent en évidence l'importance du travail d'équipe entre pédiâtre, radiologiste, bronchologiste et chirurgien thoracique dans la conduite du problème intéressant qui est discuté ici, et le fait que, lorsqu'une anomalie majeurs est découverte, il y a fréquemment d'autres malformations graves des autres systèmes qui doivent aussi être reconnues.

ZUSAMMENFASSUNG

Angeborene Entwicklungsfehler der tieferen Atemwege werden beschrieben und klassifiziert und anschliessend die klinischen Gesichtspunkte besprochen. Entwicklungsfehler der Trachea umfassen Verengerungen und Erweiterungen, Ausstülpungen und Auswüchse und abnorme Bifurkation oder Verzweigung. Entwicklungsfehler der Bronchien und der Lungen, die beschrieben werden, umfassen Aplasie, Verengerungen, Ausstülpungen, fehlende und überzählige Bronchien, Lappen und Spalten und abnormales bronchiales und Lungen-Gewebe in Verbindung mit dem Atmungsapparat oder anderen Organen. Es zeigt sich, dass die klinische Bedeutung der Entwicklungsfehler am grössten ist während der Periode des Neugeborenen, wobei die akuten respiratorischen Symptome, zu denen sie führen können, alsbaldige diagnostische Massnahmen erfordern einschliesslich der Bronchoskopie und der diagnostischen Thorakotomie. Es wird besonderer Wert gelegt auf das klinische Bild, das durch die verschiedenen Grade des Bronchialverschlusses infolge cardialer und vaskulärer Missbildungen zustandekommt. Die Besprechung betont die Bedeutung der Zusammenarbeit von Kinderarzt, Röntgenologen, Bronchologen und Thoraxchirurgen zur Erfassung der dargestellten interessierenden Probleme und weiter die Tatsache, dass, wenn eine erhebliche Anomalie gefunden worden ist, häufig noch andere schwerwiegende Missbildungen anderer Organe bestehen, die man auch diagnostizieren muss.

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The Syndrome of Relief of Angina Pectoris Following Myocardial Infarction

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Although heart disease continues to increase in frequency among the causes of death in the population of the country, and although coronary heart disease constitutes the majority of cardiac disabilities, the prognostic outlook of coronary artery disease continues to improve steadily with time. This has recently been stressed in the medical literature and has even permeated into the lay press. It has been steadily emphasized that modern diagnosis and modern therapy have lessened the mortality of each individual episode of myocardial infarction and that following myocardial infarction, many individuals return to a useful life. It has been the experience of the authors that myocardial infarction occasionally is a blessing in disguise in that it causes the cessation of anginal chest pain which had existed previously. Such a medical paradox can be explained by recently observed patho-physiological mechanisms. This knowledge has been shared by other physicians and cardiologists, but the basis for the syndrome has never been satisfactorily described in the literature.

Anatomic Considerations

The subjective manifestations of acute coronary occlusion indicate extensive sensory receptor innervations of the heart with emerging afferent nerve fibers making widespread communications with a system of efferent neurons.[1] The afferent nerve impulses come from the heart through the inferior cardiac nerves, the stellate ganglion, the cardiothoracic nerve, the thoracic sympathetic chain and the rami communicantes from the eighth cervical to the fifth thoracic segment.[2] It has also been suggested that afferent impulses from the heart are carried by the vagus nerve.[3] Many afferent impulses reach the local segmental areas of the spinal cord and produce reflex muscular spasms of the thorax. Most impulses apparently reach the hypothalamic region where they communicate with all the vegetative nuclei, especially the vagal system, which causes visceral spasms, sweating and other symptoms.

Pathology

The clinical manifestations of acute myocardial infarctions are not solely the result of acute closure of a coronary artery. In some cases, acute myocardial infarction occurs without new narrowing or occlusion, while in other cases, the acute occlusion is neutralized by collateral circulation which had formed as a result of the prior narrowing of the vessel. Thus, acute myocardial infarction is to be regarded as the resultant of two opposing processes; namely, the obstruction to blood flow by occlu-

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sive processes and the compensatory development of collateral circulation. Such collateral circulation serves to offset the dire effects of coronary artery narrowing and occlusion.[4]

At examination of the heart post mortem, only the end result of the obstructive process and compensatory mechanism can be observed. The classical experiments of Blumgart et al [5, 6, 7] have shown that collateral channels are tailored specifically to the narrowing process which evokes them. They do not develop indiscriminately, and time is required for

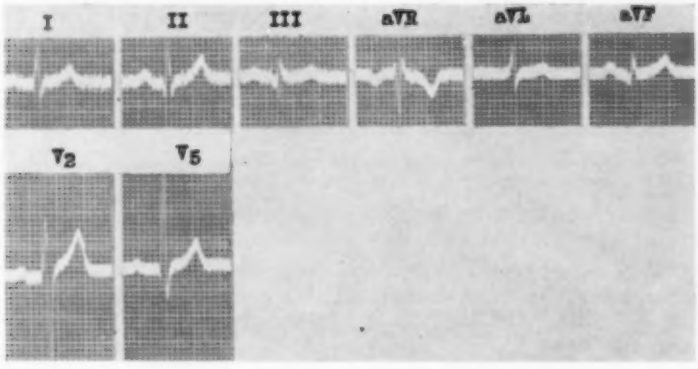

FIGURE 1 (Case 1): M. K. January 3, 1950.

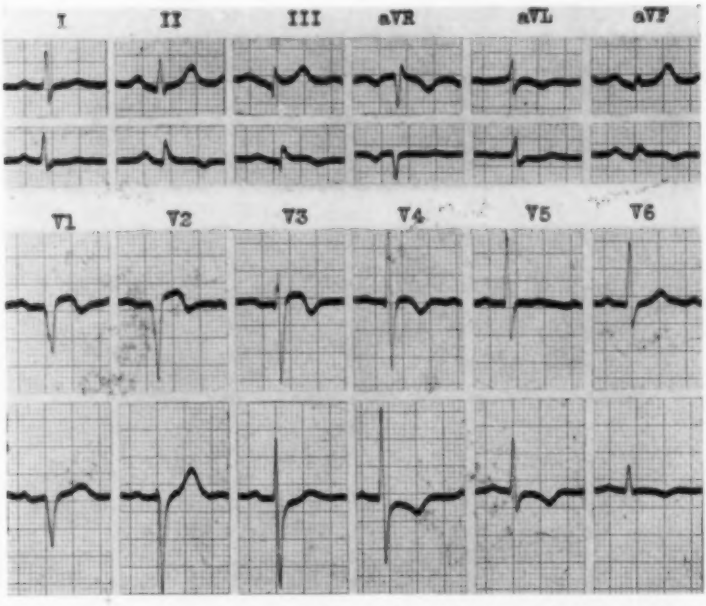

FIGURE 2 (Case 1): M. K. June 16, 1952. FIGURE 3 (Case 1): M. K. Sept. 29, 1952.

their development. Of the types of coronary arterial anastomoses which these workers have described, intercoronary anastomoses seem to be most definite and most important in man. At post mortem study, only 15 per cent of normal hearts were found to show such interarterial communications, while such communications were found in 98 per cent of hearts which had suffered coronary occlusion. In addition to these anastomoses, there are extrinsic coronary anastomoses between coronary arteries and vasa vasorum, vessels of precordium, diaphragm, mediastinum and pleura.[8] These anastomoses are of lesser importance.

The collateral channels in diseased hearts are of great functional significance. Despite complete occlusion in some hearts, the potentially infarcted myocardium has been free of fibrosis or other structural abnormality when coronary arterial narrowing progresses gradually. Complete occlusion of one or even three main coronary arteries may be compatible with continued life. The slow development of a rich anastomotic circulation may be responsible for the occasional clinical improvement of patients with angina pectoris. The collateral anastomoses act to lessen the effects of arterial narrowing with arterial occlusion when such occurs. The syndrome to be described as illustrated by the following cases evidently represents the clinical counterpart of this pathological condition.

Case Reports

Case 1: M. K. presented himself on January 3, 1950 when he was 54 years of age. Since December, 1949 he had experienced substernal pain associated with heaviness and aching of the left arm occurring with effort. This sensation was relieved by rest. He noticed that the substernal distress was worse when walking rapidly and also after a large meal. Physical examination was not remarkable and blood pressure was 124/88. X-ray film showed the heart to be of normal size. Wassermann, blood sugar, blood

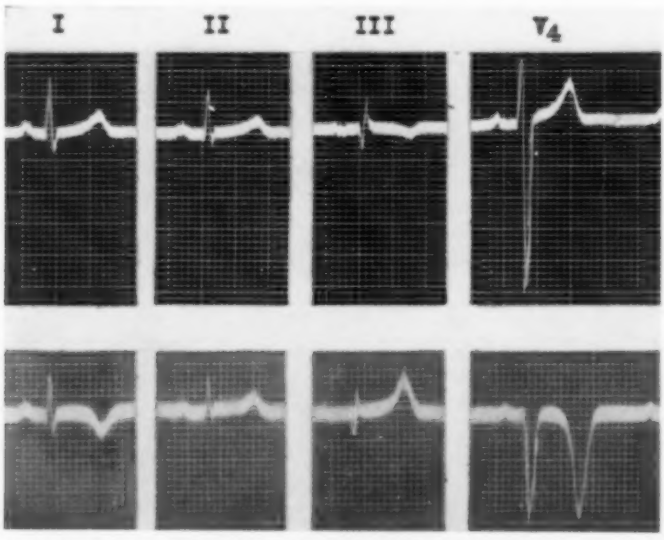

FIGURE 4 (Case 2): H. J. Jan. 10, 1940. FIGURE 5 (Case 2): H. J. July 23, 1943.

cholesterol, calcium and phosphorus were normal. Blood urea nitrogen was 17.6 mgm. per cent. Blood count and urinalysis were normal. The electrocardiogram was within normal limits (Figure 1). He was treated with vasodilators to which there was no response. There was an excellent response to nitroglycerin.

On June 15, 1952 he suffered an acute myocardial infarction for which he was treated with the usual conservative measures at home. Figure 2 shows changes of acute postero-septal wall myocardial infarction. He recovered from the myocardial infarction but soon thereafter developed severe angina. This increased in frequency and intensity so that by the beginning of September, 1952 it occurred at rest as well as with effort. Blood pressure was 120/90 and physical examination was not remarkable.

On September 29, 1952 he suffered another episode of acute coronary thrombosis with myocardial infarction which necessitated hospitalization. His clinical condition was critical and was associated with marked shock as well as congestive heart failure. Figure 3 taken on September 29, 1952 indicates acute postero-lateral wall myocardial infarction. Once he recovered from the critical phase, he did quite well, so that by December 31, 1952 he was able to work full-time. Then there was no anginal symptom and he felt quite well. Digitalis was continued. The x-ray film showed a slight increase in the transverse diameter of the heart. When last examined on September 29, 1954, when he was more than 58 years of age, he reported that he felt quite well and was able to work full time. There was no anginal symptom and the physical examination was normal. The blood pressure ranged about 110/60 mm. Hg.

Case 2: H. J. When 50 years of age, this man presented himself on January 10, 1940 for routine examination. This white executive had no cardiac symptom and examination was completely normal. The electrocardiogram was normal (Figure 4). He was next seen on June 18, 1943 when he complained of severe anginal pain which occurred both with effort and at rest. He stated that he had experienced frequent attacks of angina pectoris since using a lawn mower three months previously.

On July 1, 1943 he suffered acute myocardial infarction of the anterior wall. Figure 5 taken July 23, 1943 shows changes of anterior wall myocardial infarction. Following uneventful recovery from this episode, he remained symptomatically well and was able to perform all his work and to take part in all his social activities. He was examined yearly and remained well. The electrocardiogram showed persisting signs of old an-

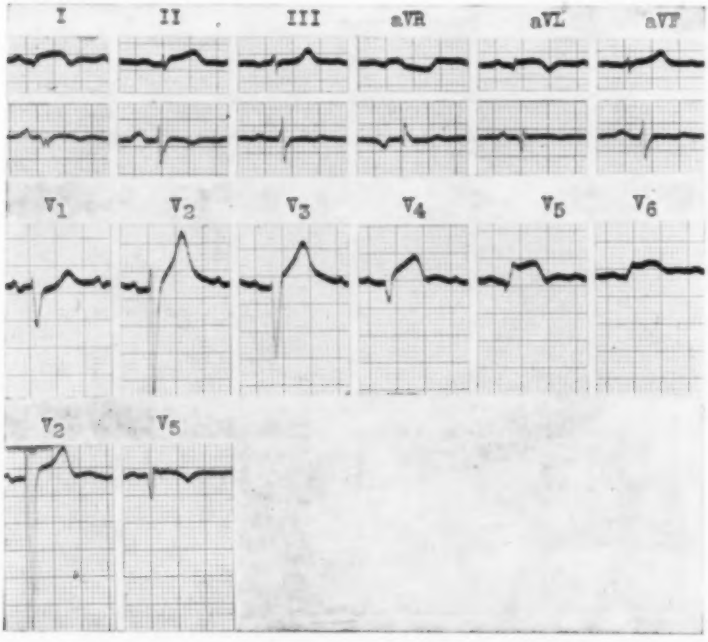

FIGURE 6 (Case 2): H. J. Jan. 15, 1953. FIGURE 7 (Case 2): H. J. Sept. 21, 1953.

terior wall myocardial infarction. When examined on October 10, 1952 he said that anginal pain had occurred during the four previous months. This pain occurred with effort and also at rest, so he was compelled to limit his physical activities.

On January 12, 1953 he suffered a second episode of acute myocardial infarction which was attended by severe shock, marked hypotension, and congestive heart failure. The electrocardiogram indicated acute antero-lateral wall myocardial infarction (Figure 6 taken January 15, 1953). Following a stormy course, he made a recovery from this episode, but mild congestive heart failure remained. When he was examined on April 29, 1953 he stated that he no longer experienced chest pain with effort or at any other time. He was completely asymptomatic, although examination showed signs of mild congestive heart failure. As of October 4, 1954 he was attending to all his

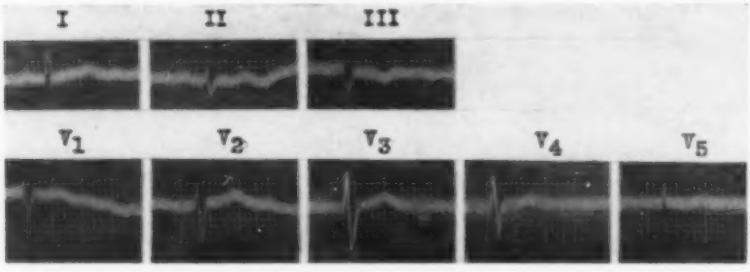

FIGURE 8 (Case 3): A. S. January 30, 1949.

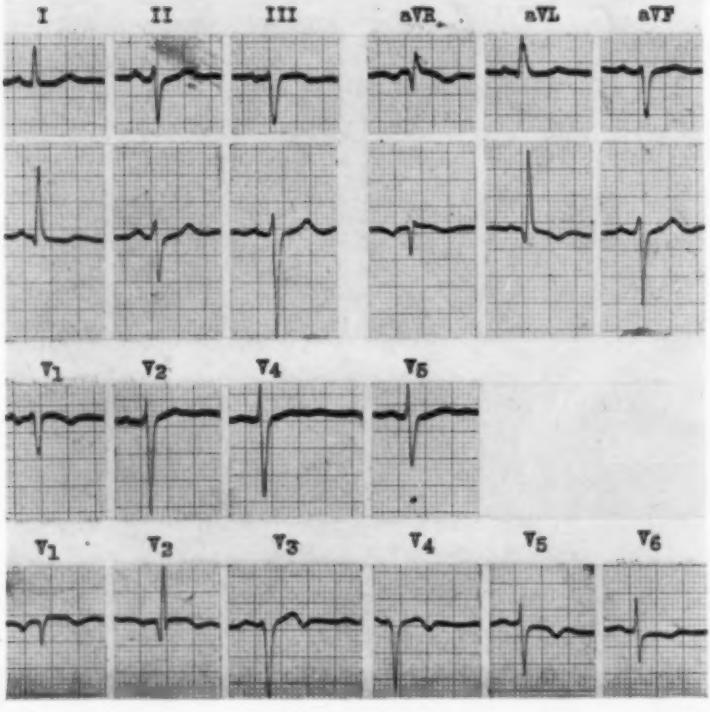

FIGURE 9 (Case 3): A. S. Sept. 11, 1953. FIGURE 10 (Case 3): A. S. May 19, 1954.

business and social functions without symptoms. The electrocardiogram taken on September 21, 1953 (Figure 7) showed the residual signs of old anterior wall myocardial infarction.

Case 3: A. S. When 52 years of age in 1949 this well developed adult white artist had been experiencing precordial pain on effort for several years. Physical examina-

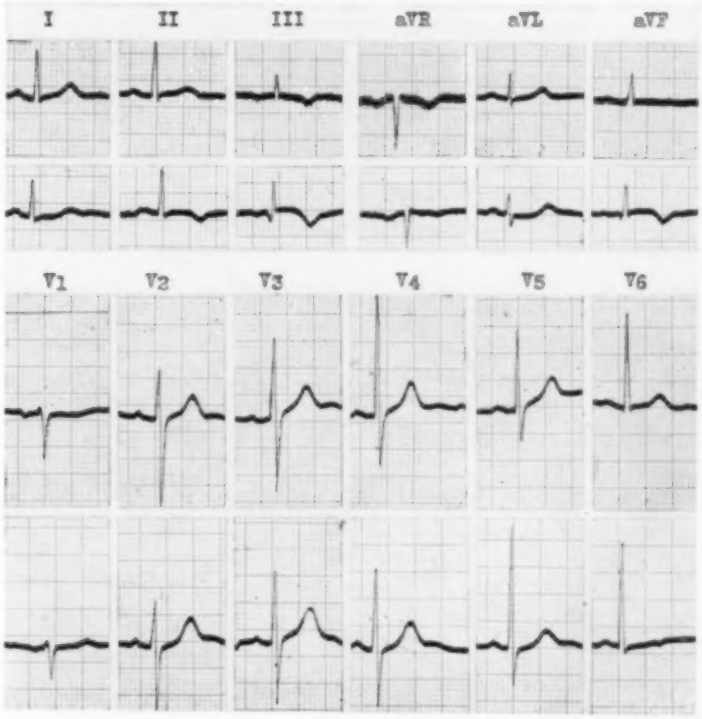

FIGURE 11 (Case 4): M. C. Feb. 3, 1951. FIGURE 12 (Case 4): M. S. Aug. 2, 1951.

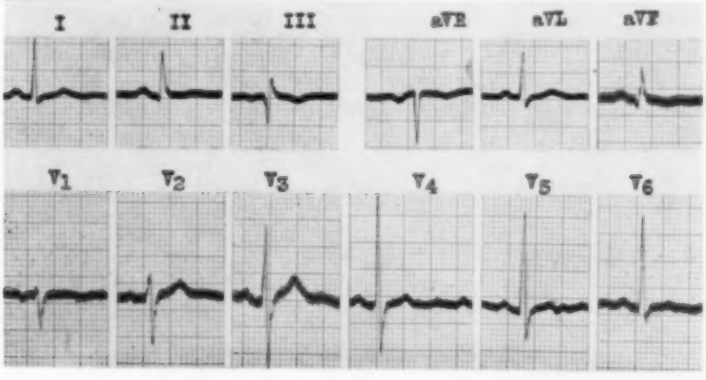

FIGURE 13 (Case 4): M. C. September 20, 1954.

tion as well as electrocardiographic study were reported as within normal limits (Figure 8). Because of the presence of gall stones, cholecystectomy was performed and it was hoped that this might help the angina pectoris syndrome. Following operation, however, there was no relief from the symptoms of angina pectoris which increased in severity and frequency. By September, 1953 he suffered from severe status anginosis so that he was confined to his room and had not attended to business in several months. Figure 9 is an electrocardiogram taken September 11, 1953. On September 20, 1953 he suffered an episode of acute myocardial infarction complicated by severe shock. He was hospitalized during this acute episode and was in critical condition during the major portion of the hospital stay. When examined on May 19, 1954 he reported that he no longer had any symptom of angina pectoris. He was able to walk without pain, drove his car and was able to attend to his business. His major difficulty at this time was a moderately severe cardiac neurosis which caused him to feel inadequate and insecure. Physical examination disclosed a rough systolic murmur at the apex and at the aortic area, blood pressure was 144/72, and x-ray film showed moderate enlargement of the left ventricle of the heart. The electrocardiogram (Figure 10) revealed evidences of old anterior wall myocardial infarction. The patient continued to be fairly well until the morning of July 2, 1955, when he was awakened from sleep by severe crushing anterior chest pain and died shortly thereafter. Death was thought to be due to a new episode of acute myocardial infarction.

Case 4: M. C. In 1951 this 43 year old salesman complained of precordial pain with effort. The pain necessitated limitation of his physical activities and of the amount of traveling he would normally do in association with his occupation. In the past, he had been treated for gall bladder disease and peptic ulcer. His brother and father had suffered coronary thrombosis early in life. Physical examination, laboratory studies, and study of heart size all were found to be within normal limits. The resting electrocardiogram (Figure 11) as well as the Master two step exercise test were found to be normal. Because of increasing frequency of chest pain with effort, he was well on his way to becoming a complete cardiac invalid when he suddenly suffered an episode of acute coronary thrombosis on August 1, 1951. Figure 12, the electrocardiogram taken on August 2, 1951, shows changes of acute posterior wall myocardial infarction. He was treated with anticoagulants at home and made an uneventful recovery. Within two months, he was able to return to his occupation as a salesman and he reported that he no longer experienced any chest pain with effort. He follows a low-fat, low-cholesterol diet with little change in his weight. He is now able to pursue his full activities and travels freely by rail, automobile, and airplane. Because of increased

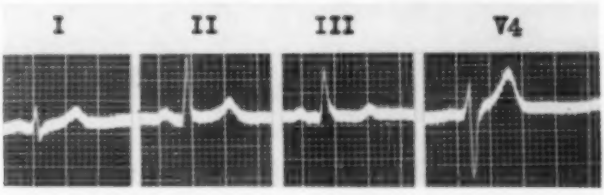

FIGURE 14 (Case 5): A. E. May 26, 1942.

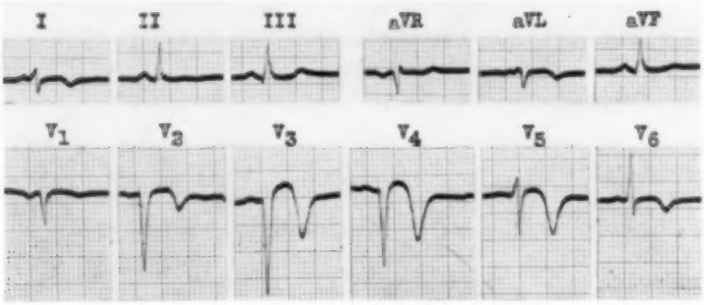

FIGURE 15 (Case 5): A. E. June 14, 1949.

endeavor, his present earning capacity is greater than that of any prior year. When examined on September 20, 1954 he appeared well, was adjusted mentally, and was happy to report that he no longer suffered chest pain. The electrocardiogram showed persisting evidence of old posterior wall myocardial infarction (Figure 13).

He neglected to return for a scheduled check-up examination, and upon inquiry it was learned that he had placed himself in the hands of a naturopath. The latter has informed him that he never suffered any cardiac injury and evidently the patient feels well enough to accept this judgment.

Case 5: A. E. This real estate executive was first observed in 1942 when he was 43 years of age. He complained of mild precordial chest pain and slight dyspnea associated with effort. Complete physical and laboratory examinations were normal. The electrocardiogram (Figure 14) taken on May 26, 1942 was normal. While the family history was positive for diabetes, studies of the patient for this disease were negative. He was observed at frequent intervals. His angina became more severe so by the end of 1948 he was forced to curtail some of his business and social activities. Throughout this interval, however, the electrocardiogram remained normal. On May 2, 1949 he suffered acute coronary thrombosis for which he was hospitalized and treated with anticoagulants. Figure 15 is the electrocardiogram taken June 14, 1949 showing changes of acute anterior wall myocardial infarction. On July 12, 1949 he reported that he was completely free of symptoms and that he was able to walk with no chest pain. With the consent of the attending physician he was permitted to return to busi-

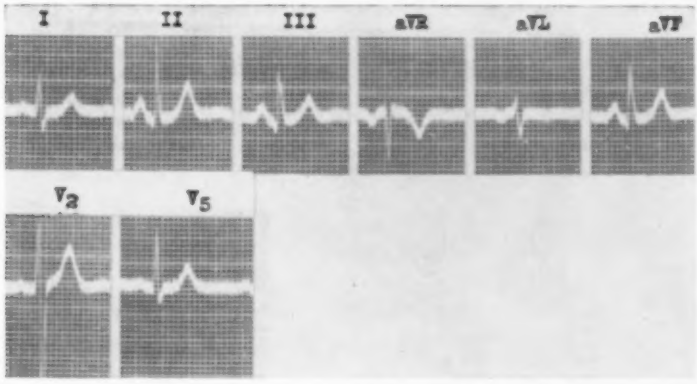

FIGURE 16 (Case 6): M. S. December 18, 1951.

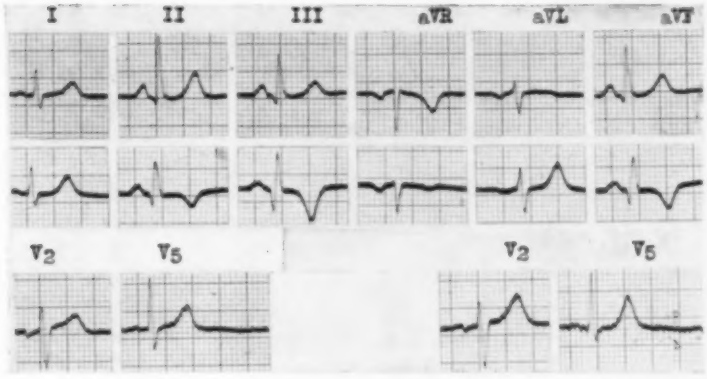

FIGURE 17 (Case 6): M. S. Feb. 20, 1953. FIGURE 18 (Case 6): M. S. Apr. 8, 1953.

ness and within a month was more active than he had been in the previous seven years. He reported regularly for examination and always stated that he was completely free of chest pain although he had undertaken additional business obligations. He continued with full business activities and was frequently observed. The electrocardiogram continued to show evidence of the old anterior wall infarction and no cardiac enlargement was recognized on fluoroscopy or x-ray film of the chest. On April 3, 1952 while walking, he suddenly collapsed and was found dead upon arrival of medical aid. He had been completely active and without symptoms until this time, some two and one half years following the acute myocardial infarction.

Case 6: S. M. This 44 year old white man presented himself on December 18, 1951 complaining of angina pectoris on effort for two and one half months. The pain had increased steadily in severity. Physical examination as well as determinations of the uric acid, cholesterol, basal metabolic rate, sedimentation rate and complete blood count, were normal. The electrocardiogram was normal (Figure 16). When seen on July 23, 1952 he was suffering from status anginosis so he was unable to attend to his business. Physical examination remained normal and the blood pressure was 125/80. The electrocardiogram remained unchanged. Examination on February 20, 1953 showed no essential change in his condition. He was virtually confined to his home. The electrocardiogram remained normal (Figure 17).

On March 21, 1953 he suffered acute coronary thrombosis for which he was treated at home. Figure 18 is the electrocardiogram taken April 8, 1953 showing evidence of posterior wall myocardial infarction. When examined on May 11, 1953 he was happy to report that he was completely without chest pain and that he had returned to his regular occupation. This entailed using the subway system and he was able to

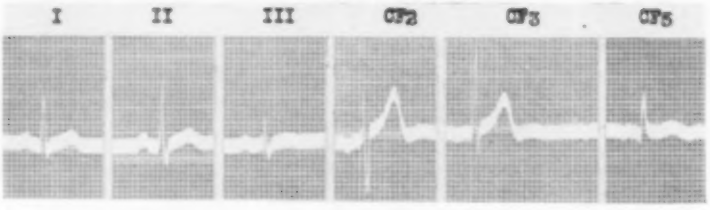

FIGURE 19 (Case 7): P. W. November 9, 1945.

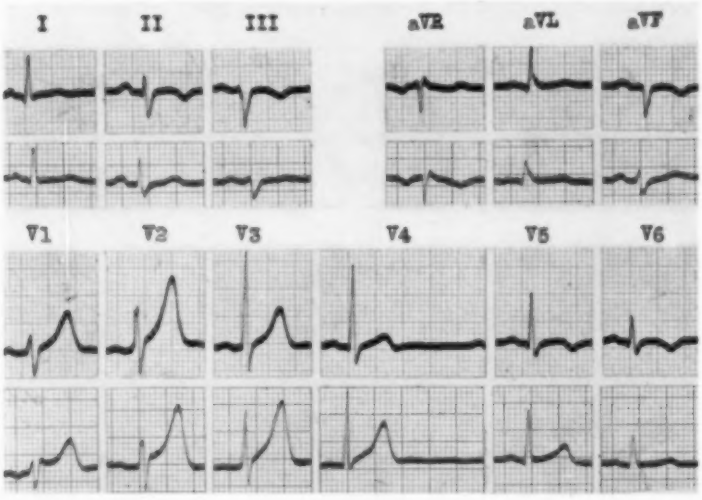

FIGURE 20 (Case 7): P. W. Oct. 4, 1954. FIGURE 21 (Case 7): P. W. March 21, 1955.

walk subway stairs without discomfort. He was now more active physically than he had been in the previous two and one half years. He continues to remain well, attends to his business and requires no active therapy.

Case 7: P. W. This 46 year old business man was observed on November 9, 1945 as part of a routine physical examination which as well as the electrocardiogram were reported as normal. Figure 19 is the electrocardiogram taken November 9, 1945. He again presented himself on September 14, 1951 stating that he had recently begun to experience substernal pain with effort and after his large meal. Walking was particularly likely to produce substernal pain. The electrocardiogram taken at this time remained normal. He was seen periodically thereafter and reported that the anginal pain increased in frequency and intensity.

On September 20, 1954, while walking up-grade, he developed intense substernal pain which was not relieved after 30 hours of rest. The diagnosis of acute coronary thrombosis was made clinically and he was hospitalized for a period of four weeks. Figure 20 is the electrocardiogram taken October 4, 1954 showing changes of posterolateral wall myocardial infarction.

On October 30, 1954 he returned to business and has since been working regularly full-time. When examined on March 21, 1955 he felt perfectly well, did not experience symptoms of angina with effort or with other activity. The electrocardiogram (Figure 21) had returned towards normal.

Comment

While it has been frequently observed that angina pectoris is experienced for the first time after an episode of myocardial infarction, the syndrome which is described illustrates that angina pectoris may be relieved following myocardial infarction. In Cases 1 and 2, the angina pectoris became more intense after the first episode of myocardial infarction, but was relieved after the second. In Case 3, the myocardial infarction afforded relief from angina pectoris which had been present for four years. There also was evidence of increased coronary reserve in that the patient was able to increase his activities greatly. However, death occurred due to a new coronary thrombosis two years later, in spite of his apparent well being. In Case 5 there was relief of chest pain after myocardial infarction. Death occurred some three years later.

Those who experience relief of angina after myocardial infarction evidently have increased functional capacity. Whether this relationship indicates a better life span remains to be studied. It would seem that one could draw a parallel between the cases described which indicate compensatory collateral circulation and the recently proposed surgical procedures which attempt to increase and promote circulation through revascularization of the heart.[9]

SUMMARY

Cases are reported to illustrate that there may be relief from severe and often intractable angina pectoris following acute myocardial infarction. While the cases described showed varying clinical manifestations and varying localization of cardiac pathology, there was the common experience of relief from chest pain following acute coronary artery occlusion. The likely explanation for the relief is that compensatory collateral circulation had developed during the period when angina pectoris was present.

In addition to the theory of increased collateral circulation following coronary artery narrowing, it has been theorized that transformation of an ischemic myocardial area into scar tissue might prevent the origin of

painful stimuli.[10] The latter theory seems less likely in view of the relatively good functional capacity after myocardial infarction in the patients described.

RESUMEN

Re relatan casos que ilustran el hecho de que puede haber alivio de una angina de pecho a veces intratable y grave al parecer un infarto del miocardio. Si bien los casos descritos mostraron variables manifestaciones clínicas y diversas localizaciones de la patología cardiaca hubo la experiencia común de alivio del dolor del pecho después de la oclusión coronaria. La explicación verosímil es que se desarrolló circulación compensadora colateral durante el período en que había angina.

Además de la teoría del aumento de la circulatión colateral después del estrechamiento coronario, se ha teorizado sobre la posibilidad de que la transformación de una área isquémica en cicatriz evite la existencia de una zona de estímulo doloroso.[10] Esta teoría parece menos plausible en vista de la capacidad funcional relativamente buena después de infarto miocárdico de los enfermos descritos.

RESUME

L'auteur rapporte des cas montrant qu'un infarctus du myocarde peut apporter un soulagement dans l'angine de poitrine sèvére et souvent incurable. Alors que les cas décrits étaient caractérisés par des manifestations cliniques diverses et des localisations variées d'atteinte cardiaque, on a pu noter généralement, après l'occlusion coronarienne aiguë, un soulagement des douleurs thoraciques antérieures. L'explication vraisemblable en est donnée par le fait que la circulation collatérale compensatrice se développe pendant la période d'angine de poitrine.

Outre la théorie de la circulation collatérale augmentée après l'étranglement de l'artére coronaire, on a pu émettre l'hypothèse que la transformation d'une zone myocardique ischémique en tissu cicatriciel pourrait empêcher l'origine des stimuli douloureux. Cette dernière théorie semble moins vraisemblable si l'on considère la capacité fonctionnelle relativement bonne après un infarctus myocardique chez les malades étudiés.

ZUSAMMENFASSUNG

Wiedergabe von Fällen, um zu erläutern, dass eine Erholung nach einer schweren und oft behandlungsrefraktären Angina pectoris im Anschluss an einen akuten Myocard-Infarkt möglich ist. Während die beschriebenen Fälle klinisch verschiedene Bilder boten mit wechselnder Lokalisation der cardialen Veränderungen, bot sich ganz allgemein die Erfahrung einer Erholung von Brustschmerzen im Anschluss an einen akuten Coronar-Arterien-Verschluss. Die wahrscheinliche Erklärung für die Erholung besteht darin, dass es zur Entwicklung eines kompensatorischen Kollateralkreislaufes gekommen ist während des Zeitabschnittes, in dem die Angina pectoris vorlag.

Zusätlich zu der Theorie des erhöhten Collateral-Kreislaufes im Anschkuss an eine Verengung der Coronar-Arterien wurde theoretisch erwogen,

dass die Umwandlung eines ischaemischen Myocard-Bezirkes in ein Narbengewebe das Auftreten schmerzhafter Reize verhindern könnte.[10] Die letzgenannte Theorie scheint wenig wahrscheinlich im Hinblick auf die relativ gute funktionelle Leistung der beschriebenen Fälle mit Herzmuskelinfarkt.

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Simultaneous Mantoux and Vollmer Patch Tests in 855 School Children*

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Los Angeles, California

In March 1955, a comparative study of the results of the (Mantoux) intracutaneous tuberculin test, and the patch test, (Vollmer) was carried on in two Los Angeles schools. The purpose of this study was to determine whether or not the patch should be substituted for the intracutanous test, allowing greater ease of administration, decreased expense, and lessened work load.

The schools chosen were two junior high schools where the students ranged in age from 11 to 14 years. The economic level of the students was, in general, below average for the City as a whole. While many races were represented in the group of those tested, the majority were of the Negro race. A total of 1,049 pupils received both tests simultaneously, the Mantoux test being given on one forearm, the patch test on the other. Of this number, 194 were absent for interpretation of the tests. In another group of 180 students the patch test became detached before the 48-hour interval at the end of which it should have been removed. Of these students who "lost" their patch tests, 25 had positive Mantoux tests and 155 had negative Mantoux tests. The number tested and inspected 48, 72, and 98 hours after removal of the patch test was 855; the results in these students form the basis of this study.

Technique of Study

The Mantoux test used was 0.00025 mg. of PPD, or 12.5 tuberculin units. The test solution used was made by dissolving tablets of PPD in normal saline solution within two hours before injection. The student's left forearm was cleansed with alcohol. Using a 26 gauge, 3/8 inch steel needle and a tuberculin syringe, 0.1 ml. of solution containing 0.00025 mg. of PPD was injected intracutaneously. The needles were flamed in an alcohol lamp flame between patients and allowed to cool before being reused. The tests were given by one of the three authors (W. J. R.) and great care was taken to measure the amount injected intradermally exactly to 0.1 ml. and to produce a skin wheal.

The interpretation of the Mantoux test in each case was done 48 hours after it was given, by a team of two nurses, both of whom are experienced in such work as they are regularly assigned full time to the tuberculin testing program in the Los Angeles City Schools. The tests were interpreted according to the diagnostic standards of the National Tuberculosis Association, with 1 plus being an area of edema and redness between 5 mm. and 10 mm. in diameter; a 2 plus between 10 mm. and 20 mm. in

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diameter; a 3 plus exceeding 20 mm. in diameter; and a 4 plus showing evidence of necrosis.

The tuberculin patch tests were supplied for the study by one of the leading drug concerns. The strength of each patch test has been stated to be equivalent to 2.5 tuberculin units. A representative of the manufacturer was present to observe that it was properly applied. The right forearm was cleansed with acetone, which was then allowed to dry thoroughly. While the doctor was administering the Mantoux test to the inner surface of the left forearm, the adhesive guard was removed from the patch test by a nurse and the patch firmly applied transversely to the inner or flexor surface of the right forearm. The student was instructed to hold it firmly for five minutes until the heat of the hand had allowed it to adhere. The students were instructed that they were not to remove the patch until in the presence of the testing nurse 48 hours later.

Forty-eight hours after *application* of the Mantoux and patch tests, the students assembled and one at a time were checked by a team of two nurses who removed the patch test and recorded the reading of the Mantoux test.

A day later the students were again assembled and the patch test inspected, a record being made if it was positive at this time. Again a day later, or 48 hours after the patch had been *removed* (as recommended by the maker of the tuberculin patch tests) the area was inspected and the reading of the patch test recorded. The criterion for interpretation of the patch test as positive was the presence of one or more papules in the area under the test substance areas, with no such reaction under the control area.

Results of the Study

As is shown in Table I, of a total of 196 positive Mantoux readings, 83 had positive patch tests. The tests agreed in 739 cases (83 + 656) and disagreed in 116 cases (113 + 3). Probability tests indicate that this difference is highly significant.

The positive readings of the Mantoux test were further broken down into the degree of reaction as shown in Table II. While there is a high degree of correlation between cases in which skin sensitivity was high, and Mantoux readings were three or four plus, the majority of the cases where the Mantoux reading was one or two plus had negative patch tests.

TABLE I

MANTOUX

		+	—	Total
Patch Test	+	83	3	86
	—	113	656	769
	TOTAL	196	659	855

This difference in reading between the two testing methods is more effectively shown in Figure 1.

Discussion

In considering this difference between the results of the patch and the intracutaneous methods, one must note that the dosage of tuberculin units varies in the two tests—that of the patch is stated to be 2.5 tuberculin units and that used in the intracutaneous is stated to be 12.5 tuberculin units. This latter dosage is two and one-half times the standard intermediate strength of purified protein derivative (.0001 mg. per dose or five tuberculin units). For several years the 5-tuberculin-unit test had been used in the Los Angeles City schools, but because of the large number of doubtful reactions (with induration less than 5 mm.) the strength was raised to .00025 mg. per dose and far fewer doubtful reactions have been noted. This increased dosage does not make for any wholesale production of positive reactions as these findings show a graded distribution of degrees of positivity, the most weight centering at the minimal reaction end of the scale.

It is of interest that Aronson, Taylor, and McGettigan[1] point out that reactions in individuals vaccinated with BCG often fall into the doubtful area, with only slight induration, and largely consist of erythema. Such reactions are considered significant inasmuch as such individuals had absolutely negative reactions prior to vaccination. These authors point out that tuberculin hypersensitivity in BCG vaccinated adults is usually of low grade, and increasing the dose of tuberculin does not appreciably affect the degree of the skin reaction.

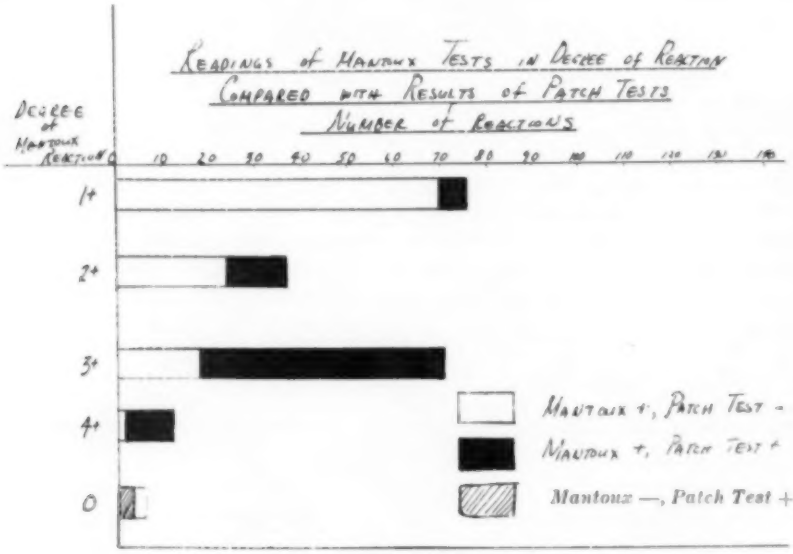

FIGURE 1

TABLE II

INTERPRETATION OF DEGREE OF REACTION OF INTRACUTANEOUS TEST WITH RESULTS OF PATCH TESTS

Intracutaneous Reading	1+	2+	3+	4+	Total
Intracutaneous positive Patch test positive	6	13	53	11	83
Intracutaneous positive Patch test negative	70	24	18	1	113

On the other hand, Palmer and Nash[2] feel that a dose of five tuberculin units is the one most likely to give a distinction between non-specific reactors and those with true tuberculin hypersensitivity, especially if a positive is read at eight mm. induration or above.

Another point to be considered is the importance of readings of one and two plus Mantoux reactions. It has been reported by Hetherington, McPedron, Landis and Opie,[3] Wells and Smith,[4] Aronson and Aronson[5] that significant lesions found roentgenographically react definitely to small initial dosage of various tuberculins.

A review was made of the active cases, with the diagnosis based on positive bacteriological findings, reported in the Los Angeles City schools in the past few years where the tuberculin test of the intermediate dose of .00025 mg. (12.5 tuberculin units) was given and interpreted just as in this study. In fact these cases were diagnosed because just such a positive test initiated a diagnostic follow-up. It was found that of a total of 45 such cases, nine Mantoux tests had been interpreted as one plus, 11 as two plus, 15 as three plus and 10 as four plus. This small series would indicate that a reading of one or two plus holds definite significance when this tuberculin dosage is used.

Many studies have shown a fairly close correlation between the patch test and intracutaneous tests. Vollmer[10] reported his patch test to be more effective than the Pirquet method; and in a study of 169 tuberculous children, the patch test (using a concentrated preparation of O.T.) revealed only one less positive reactor than with first and second strength of PPD.

Krug and Glenn[6] in a study of 2,152 college students found 22.1 per cent positive reactions to first and second strengths of PPD, and 20.2 per cent positive reactions to the patch test. Of 2,401 infants who were given BCG intracutaneously 24 to 48 hours after birth and tested two months to two years later, 98.4 per cent gave a positive patch reaction. Tuberculin hypersensitivity has been noted to be high in infants as compared to adults following BCG. Savage[7] tested 437 persons and found a fairly close correlation with 39.8 per cent positive patch reactions, 46.5 per cent reactors to .01 mg. O.T., and 1 mg. O.T., and 47.6 per cent positives using first and second strengths PPD.

Baldwin[8] in a study of West African natives found that 58 per cent

reacted to the patch and .01 mg. of O.T., 31.6 per cent showed negative reactions to both tests and slightly more than 10 per cent showed disagreement with the majority giving a positive intracutaneous and a negative patch. Again the intracutaneous was shown to be only slightly more effective in revealing positive reactions than the patch test. Quintos and Santiago[9] state that the patch and .01 mg. O.T. are about equally effective in demonstrating positive reactors, more so than .00002 mg. (1 tuberculin unit) of PPD, and 16.6 per cent less effective than 1 mg. O.T.

There have been, however, studies that indicate that the patch is less effective than the intracutaneous test.

Aronson, Taylor and McGettigan tested 123 adult males who received BCG two months previously. One hundred and eighteen or 95.9 per cent reacted to .01 mg. O.T. while (51.2 per cent) or 63 reacted to the patch test. These workers also tested 88 cases, who did not receive BCG, simultaneously with the patch and intracutaneous test. One tuberculin unit of PPD or first strength showed 65.2 per cent reactors while 50.3 per cent reacted to the patch. Eighty-four were again tested with the patch and .01 mg. O.T. used in manufacture of the patch tests. Again, the patch test showed only 48.9 per cent positives and the intracutaneous 63.0 per cent positives.

SUMMARY

With the decreasing morbidity and mortality of tuberculosis, the tuberculin skin test assumes increasing importance. Accurate knowledge of the presence of tuberculin hypersensitivity is essential for those who desire to treat recent converters or to administer BCG. Many scientific, industrial and medical legal problems demand accurate evaluation of tuberculin hypersensitivity. Determination of accurate tuberculin hypersensitivity in younger age groups is especially valuable for diagnostic purposes in view of the large percentage of negative reactions expected.

A study was made in two Los Angeles City schools, in an effort to determine whether or not the patch test (Vollmer) could be substituted for the intracutaneous test (Mantoux). It was felt that the patch test might have advantages in the ease of administration and in acceptability to the student and his parents. The two tests were given simultaneously, using an intracutaneous test (Mantoux) of 0.00025 mg. PPD (equal to 12.5 TU) and a patch test (Vollmer) (probably equal to 2.5 TU). The skin test suffered the disadvantage in that in about 10 per cent of the tests the patch became detached before the proper time interval had lapsed. Of 855 students so tested and included in this study, 83 reacted to both tests; 113 to the intracutaneous test but not to the patch test; and 3 reacted to the patch test but not to the intracutaneous test. In general, it was noted that where the intracutaneous reaction was markedly positive, the patch test was also usually positive, but where the intracutaneous reaction was less marked, the patch test did not cause a reaction. When the patch test is positive, the intracutaneous is almost certain to confirm tuberculin sensitivity.

The group of intracutaneous positives of minimal readings (one plus

and two plus) holds special significance. If the individuals in this group had been given only the patch test many would have gone unrecognized as being sensitive. The authors acknowledge that in a clinical situation dealing with individual contact patients where higher tuberculin dosages could be used in repeat testing to rule out false negatives, the patch test is satisfactory. They are not convinced, however, that a high proportion of clinicians routinely follow a negative patch test with intradermal tests of higher tuberculin strength to exclude the possibility of a false negative reaction.

The group of intracutaneous positives of minimal readings, moreover, is apt to be productive of active cases. The authors maintain that the testing situation described herein is comparable with the previous screening in Los Angeles schools where 20 active cases with prior intracutaneous minimal readings (one plus and two plus) were determined out of 45 active cases with prior readings of all degrees.

Any failure to detect active cases far less pronounced than is indicated above would invalidate the use of the patch test in spite of this test's greater economy as to expense and work load. It is concluded, therefore, that intracutaneous tuberculin testing should remain the method of choice in Los Angeles City schools. It is acknowledged that in other testing situations the patch test may be a useful procedure.

RESUMEN

Con el decrecimiento de la morbilidad y la mortalidad de la tuberculosis, la reacción tuberculínica asume mayor importancia. Es esencial el exacto conocimiento de la presencia de hiperreactividad a la tuberculina para los que deséen tratar a los recién virados o administrar BCG. Muchos problemas, científicos, industriales, y médico-legales requieren una valuación exacta de la sensibilidad a la tuberculina. La determinación de esta sensibilidad en los grupos jóvenes es especialmente valiosa para el diagnóstico en vista del elevado porcentaje de negativos que es de esperarse.

Se hizo un estudio en dos de las escuelas de Los Angeles, para determinar si el parche de Vollmer podría substituir a la reacción intracutánea de Mantoux. Se cree que la reacción del parche tiene ventajas por su facilidad de aplicación y su fácil aceptación por los parientes y el estudiante mismo.

Las dos pruebas se hicieron simultáneamente usando para la intracutánea (Mantoux) 0.00025 mgrs. de PPD equivalente a 12.5 de TU y un parche de Vollmer probablemente equivalente de 2.5 TU. La reacción del parche tuvo la desventaja de que probablemente el 10 por ciento de esos parches se desprendieron antes de transcurrir el tiempo adecuado para quitarlos.

De 855 estudiados así, 83 reaccionaron a ambas pruebas; 113 reaccionaron a la reacción intracutánea pero no al parche; y 3 reaccionaron al parche y no a la intracutánea.

Se notó que en general cuando la reacción intracutánea era marcadamente positiva, también lo era al parche, pero cuando la intracutánea era menos marcada, el parche no daba reacción.

Cuando la prueba del parche es positiva, la reacción intracutánea casi seguramente confirma la sensibilidad tuberculínica.

El grupo de intracutáneos positivos con lecturas mínimas (de una y dos cruces) tiene especial significación. Si a estos individuos se hubiese hecho sólo la reacción del parche, muchos sensibilizados hubieran pasado inadvertidos.

Los autores reconocen que en una situación clínica tratándose de contactos de enfermos donde mayores dosis de tuberculina pudiesen ser usadas para descartar los negativos falsos, la prueba del parche es satisfactoria.

No están convencidos sin embargo, de que un número grande de clínicos hagan seguir habitualmente al parche negativo una reacción intradérmica de mayor concentración para excluir esos falsos negativos.

El grupo de positivos intracutáneos con lectura de mínimos más aún es capaz de producir casos activos. Los autores sostienen la situación de pruebas descrita aquí es comparable a las otras investigaciones en las escuelas de Los Angeles en las que 20 casos con lecturas intracutáneas mínimas (una o dos cruces) fueron encontradas entre 45 casos activos con lecturas anteriores de todas las intensidades.

Una falta de descubrir casos activos más marcada que lo indicado antes, invalida el uso del parche a pesar de su economía en gastos y trabajo. Se concluye por tanto, la reacción intracutánea de tuberculina debe permanecer como el método de elección en las escuelas de Los Angeles. Se reconoce que en otras condiciones de investigación la prueba del parche puede ser útil.

RESUME

A mesure que s'abaissent les taux de morbidité et de mortalité tuberculeuse, le test cutané tuberculinique devient de plus en plus important. Il est essentiel d'avoir une connaissance précise de la sensibilité tuberculinique quand on désire traiter ceux qui viennent de virer leurs réactions, ou administrer le B.C.G. Beaucoup de problémes scientifiques, industriels et médico-sociaux demandent l'estimation précise de la sensibilité à la tuberculine. Sa détermination chez les groupes de jeunes est particulièrement heureuse pour aider le diagnostic, lorsqu'on est dans une ambiance qui comporte un nombre important de réactions négatives.

Une étude a été faite dans deux écoles de Los Angeles pour essayer de déterminer si le timbre de Vollmer pouvait être ou non substitué au test intracutané de Mantoux. Les auteurs constatèrent que le timbre pourrait avoir des avantages en ce qui concerne la facilité de l'administration et son acceptation par les étudiants et leurs parents. Les deux tests furent faits simultanément, avec l'intra-dermo-réaction (Mantoux) de 0,00025 mgr. PPD (égale à 12,5 U.T.) et un timbre de Vollmer (probablement égal à 2,5 U.T.)

Le timbre dans environ 10% des tests eut l'inconvénient de se détacher avant que l'intervalle de temps nécessaire avant son enlévement ne se soit écoulé. Parmi 855 étudiants ainsi contrôlés, et compris dans cette étude, 83 réagirent aux deux tests à la fois. 113 réagirent à l'intradermo-

réaction mais pas au timbre et 3 réagirent au timbre mais pas à l'intradermo-réaction. En général on nota que là ou l'intra-dermo-réaction était fortement positive, le timbre l'était habituellement aussi, mais que là ou l'intradermo-réaction était moins marquée, le timbre ne provoquait aucune réaction. Lorsque le timbre est positif, il est presque certain que l'intradermo-réaction confirme la sensibilité à la tuberculine.

Le groupe des intradermo-réactions positives, de lecture douteuse (une croix ou deux croix) prend une valeur spéciale. Si les individus de ce groupe n'avaient eu qu'un timbre, beaucoup n'auraient pas été reconnus sensibles. Les auteurs pensent que dans un cas clinique concordant avec un contact connu, où les plus hauts dosages à la tuberculine devraient être utilisés de façon répétée pour dépister les réactions faussement négatives, le timbre est satisfaisant. Ils craignent toutefois qu'une proportion élevée de cliniciens ne fassent pas suivre systématiquement les timbres négatifs par des tests intradermiques, à plus fortes concentrations de tuberculine, afin d'exclure la possibilité d'une réaction faussement négative.

ZUSAMMENFASSUNG

Mit der abnehmenden Morbidität und Motalität an Tuberkulose gewinnt der Tuberkulin-Hauttest zunehmend an Bedeutung. Genaue Kenntnis des Vorliegens der Tuberkulin-Empfindlichkeit ist für alle diejenigen wesentlich, die frische Convertoren zu behandeln wünschen oder mit BCG arbeiten. Viele wissenschaftliche, industrielle und gesundheitsgesetzgeberische Probleme verlangen eine genaue Bestimmung der Tuberkulin-Empfindlichkeit.

Bestimmung der genauen Tuberkulin-Empfindlichkeit in jüngeren Altersgruppen ist von besonderem Wert für diagnostische Zwecke im Hinblick auf den hohen Prozentsatz zu erwartender negativer Reaktionen.

In 2 städtischen Schulen von Los Angeles wurde eine Untersuchung vorgenommen mit dem Ziel, ob der Pflastertest (Vollmer) anstelle des intracutanen Testes (Mantoux) treten könne. Es bestand die Auffassung, dass der Pflastertestin der Hinsicht Vorteile haben könne, dass er leicht zu verarbeiten und für die Schüler und ihre Eltern annehmbar sei. Beide Proben wurden simultan verabfolgt unter Benutzung eines intracutanen Testes (Mantoux) von 0,00025 mg. PPD (entsprechend 12,5 Tuberkulineinheiten) und ein Pflastertest (Vollmer) (wahrscheinlich entsprechend 2,5 Tuberkulin-Einheiten). Der Hauttest litt unter dem Nachteil, dass in ungefähr 10% der Proben das Pflaster entfernt wurde bevor der entsprechende Zeitabschnitt für die Entfernung verstrichen war. Von 855 so geprüften und in dieser Untersuchung aufgenommenen Schülern reagierten 83 auf beide Proben; 113 auf die Intracutan-Probe, aber nicht auf die Pflasterprobe; und 3 reagierten auf die Pflasterprobe, aber nicht auf die Intracutan-Probe. Im all gemeinen war festbustellen, dass wenn die intracutane Reaktion ausgesprochen positiv war, die Pflasterprobe auch gewöhnlich positiv ausfiel; aber wenn die intracutane Reaktion weniger ausgeprägt war, führte die Pflasterprobe nicht zu einer Reaktion. Ist die Pflasterprobe

positiv, kann man fast immer sicher sein, dass die intracutane Probe die Tuberkulin-Empfindlichkeit bestätigt.

Die Gruppe der intracutan positiv Reagierenden mit minimaler Reaktion (1+ und 2+) verlangt besondere Aufmerksamkeit. Wäre den Personen in dieser Gruppe nur der Pflaster-Test gegeben worden, wären viele nicht als empfindlich erkannt worden. Die Verfasser geben zu, dass in einer klinischen Situation, die mit Kontaktfällen im Einzelnen zu tun hat, wo höhere Tuberkulin-Dosen zur Anwendung kommen können bie grössere Zahl von Klinikern systematisch einer negativen Pflasterprobe Wiederholten Proben, um falsche negative Befunde auszuschliessen, die Pflasterprobe befriedigend ist. Sie sind jedoch nicht überzeugt, dass eine eine Intracutanprobe und höherer Tuberkulin-Menge folgen lassen, um die Möglichkeit falscher negativer Reaktionen auszuschliessen.

Die Gruppe der intracutanen positiven, aber minimalen Reaktionen ist imstande, aktive Fälle ans Licht zu bringen. Die erfasser vertreten die Auffassung, dass die hier beschriebene Situation der Hautproben vergleichbar ist einer vorausgegangenen Suchaktion in den Schulen von Los Angeles, wo 20 aktive Fälle mit früheren intracutanen minimalen Reaktionen (1+ und 2+) herausgefunden wurden unter 45 aktiven Fällen mit früheren Hautproben aller Stärken.

Jeder Versager bei der Entdeckung aktiver Fälle, der weit weniger ausgesprochen ist als oben ausgeführt, würde dem Gebrauch der Pflasterprobe abträglich sein trotz der grösseren Wirtschaftlichkeit dieser Probe hinsichtlich der Kosten und der Arbeit. Es wird daher der Schluss gezogen, dass die intracutane Tuberkulin-Prüfung auch weiterhin die Methode der Wahl in den Schulen von Los Angeles bleiben muss.

Es wird anerkannt, dass bei anderen Voraussetzungen für Hautempfindlichkeitsprüfungen die Pflasterprobe eine nützliche Methode sein kann.

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Unilateral Positive Pressure Breathing as a Means of Partial Shift of the Pulmonary Blood Flow [1, 2]

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Introduction

With the increased employment of irreversible collapse therapy and of pulmonary resection in the treatment of pulmonary tuberculosis, it becomes more important to predict the functional value of the remaining parenchyma. These problems have stimulated the development of complicated techniques for the study of pulmonary function with the aim to produce preoperatively the exact alterations of pulmonary and circulatory function which will occur after the proposed surgical intervention.

Bronchospirometry[12] enables the quantitative determination of the respiratory function of each lung separately. Comparative studies between this technique and angiopneumography[7, 14, 16, 17, 8, 20] have demonstrated that in each stabilized pulmonary pathological process a local increase of the vascular resistance takes place, with the parallel reduction of the local circulation and of the oxygen absorption; therefore bronchospirometry also gives information on the value of the blood flow distribution between the two lungs.

On the other hand angiopneumography permits a good evaluation of the pulmonary irrigation, gives us data about the function not only of each lung separately but also of each pulmonary segment.[7, 14, 16, 17, 8, 20]

Both techniques give us information about the *actual value* of the function of each lung in its two aspects, the respiratory and the circulatory, but do not permit any prevision of the *potential value* of each of them, i.e. up to what degree one lung is able to take over the function of the other, and insure by itself the oxygen need of the patient.

Selective angiopneumography[4] identifies the actual or potential pulmonary circuits, but gives no information about the influence of blood quantity which flows through the same circuits, according to their own resistance and to the pulmonary arterial pressure. For this reason it cannot show the *actual functional value* of the area under study.

The blockade of one main bronchus by means of a catheter with a rubber cuff,[1] or its connexion with a nitrogen atmosphere[13, 19, 14, 15, 17, 36] suppress the function of the corresponding parenchyma and tries to solve "grosso modo" the problem of the *potential value* of the opposite lung. Both techniques have the fault that the areas excluded from their function may maintain a good irrigation[14, 15, 17, 3, 9, 2, 10] for a long time. It

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2 Presented before the Sessions of the Sociedad Uruguaya de Tisiología y Enfermedades del Tórax, August 2 and November 1, 1955.

is not therefore possible to establish whether, in the mentioned conditions, the lowered arterial oxygen saturation is due to a blood shunt or to a deficient capacity for oxygen absorption which may be present in the normally breathing lung.

The temporary unilateral occlusion of one trunk of the pulmonary artery,[6, 18, 11, 5] by means of a small rubber cuff at the tip of a cardiac double lumen catheter, is up to now the best method of deviating the blood flow from one lung to the other. This technique permits correct estimate of the *potential functional value* of the opposite lung and direct study of the effect of reduction of the pulmonary vascular bed on the right heart.

The object of this work is to describe a method which, with a minimum of trouble and danger for the patient, permits the deviation of a great amount of the blood flow from a lung or a lobe towards the remaining pulmonary parenchyma. This circulatory exclusion of a certain segment can be maintained for a time sufficiently long to permit estimate of the *potential functional value* of the remaining parenchyma. This technique is based on the fact that the increase of the alveolar pressure in a section of the lung produces a compression in the corresponding vessels, with an increase of the local circulatory resistance.

Method and Material

After laryngo-tracheal anesthesia with 5 per cent cocaine and 2 per cent xylocaine solutions, a urethral catheter of the Foley type, from No. 18 to No. 22, 48 cm. long (made opaque to x-rays in its distal end by means of a small piece of lead) is introduced in a main or lobar bronchus. Once the correct position of the catheter has been verified by fluoroscopy, the cuff (whose capacity is 5 cm.^{3}) is inflated with air until the bronchus is completely isolated. The proximal end of the catheter is then connected with the device (Figure 1), whose parts are: a glass tube of 10 mm. interior diameter, a pressurizing unit (A) (an air compressor or a tank containing air, oxygen or nitrogen under pressure), a rubber bulb (B) for the stabilization of the pressure in the system, and a water valve (C) of variable resistance.

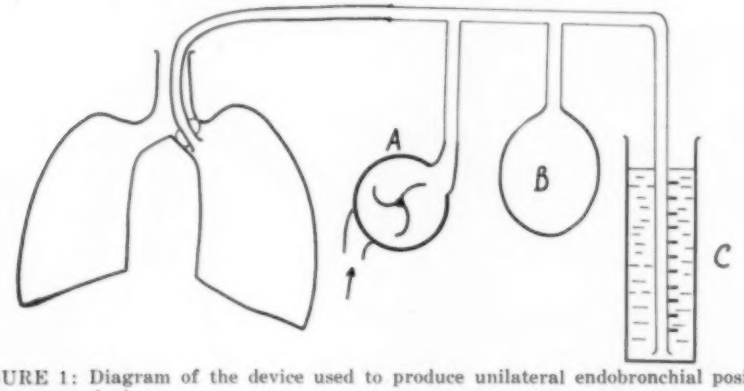

FIGURE 1: Diagram of the device used to produce unilateral endobronchial positive pressure; see text.

With the patient in supine position, the pressure in the bronchus is increased under fluoroscopic observation, in order to avoid excessive displacement of the mediastinum. With pressures between +20 and +25 cm. of water one can notice a slight displacement of the mediastinum, a lowering of the half of the diaphragm in which the mobility is reduced, increased clearness in the half of the thorax to which the positive pressure is applied, with reduction of the transparency in the opposite half.

Under these conditions, an angiopneumography was made by injecting in one of the veins of the elbow 40 cm. of a 75 per cent solution of an iodine compound. A hand operated horizontal seriograph was used which permitted four exposures at intervals of one second. Tests were made in 10 patients: four normal, two with slight unilateral residual fibrous lesions, and four with unilateral pulmonary or pleuro-pulmonary active tuberculosis. In the six pathological cases we always tried to deviate the blood towards the sick side.

Tests were made without important disturbances for the patients and without complication. At least in rest conditions, under which these tests were made, the patients did not show respiratory difficulty and it was only possible to detect a slight acceleration of the pulse and of the respiratory rhythm.

As a complementary work, 40 subjects (6 normal and 34 with unilateral or bilateral moderately advanced active pulmonary tuberculosis) were studied following standard bronchospirometry in the sitting position. We then studied the changes in the spirometric tracing of a lung breathing oxygen at atmospheric pressure while the other lung was ventilated in open circuit with nitrogen at atmospheric or positive pressure or with oxygen at positive pressure by means of the device shown in Figure 1. With this aim, we used Gebauer's catheters* and double bronchospirometer, manufactured by McKesson, Inc. We have made tracings of the tolerance of the patients to the unilateral positive pressure test, and of the variations of the respiratory rhythm, of the minute oxygen uptake (Cons. O_2), of the respiratory minute volume (V.R.M.) and of the ventilatory or respiratory equivalent (E.R.). The unilateral positive pressure was many times applied alternatively to each lung. No deleterious effect was noted in this group of patients.

Results and Discussion

Figure 2 shows the series of four films obtained in a normal woman (22 years old), whose left main bronchus had been connected to a +13 cm. of water atmosphere of air. The opaque end of the catheter can be seen in the left main bronchus. There is a slight lowering of the left half of the diaphragm without deviation of the mediastinum. In the left half of the thorax can be observed a great increase in clearness because of the increased amount of air and the corresponding reduction of the capillary bed. There exists, besides, a lack of simultaneous development of opacity

*This catheter offers a great resistance to the air flow and does not permit a prolonged testing; we were not in possession of a Carlens catheter, with which we would have sufficiently overcome this inconvenience.

in the two pulmonary arteries. In films 1 and 2 the right pulmonary tree appears in its full contrast, with the typical mottling of the capillary circulation, while the left one shows less contrast limited to the principal arterial branches. Films 3 and 4 show a marked predominance of the venous tree on the right side, while the arterial filling in the left side still remains.

Figure 3 shows four films obtained in a normal woman (38 years old), whose intermediate right bronchus was maintained under positive pressure of 25 cm. of water with nitrogen (see the opaque end of the catheter in film 1). The right half of the thorax appears visibly distended, with moderate shift of the inferior mediastinum, lowering of the right half of the diaphragm and enlargement of the inferior intercostal spaces. One can notice an increase in the clearness, caused by the increased amount of air and reduction of the pulmonary capillary bed. The development of opacity in the pulmonary arteries of the two areas is not synchronous. In fact, the arteries that correspond to the right inferior and middle lobes

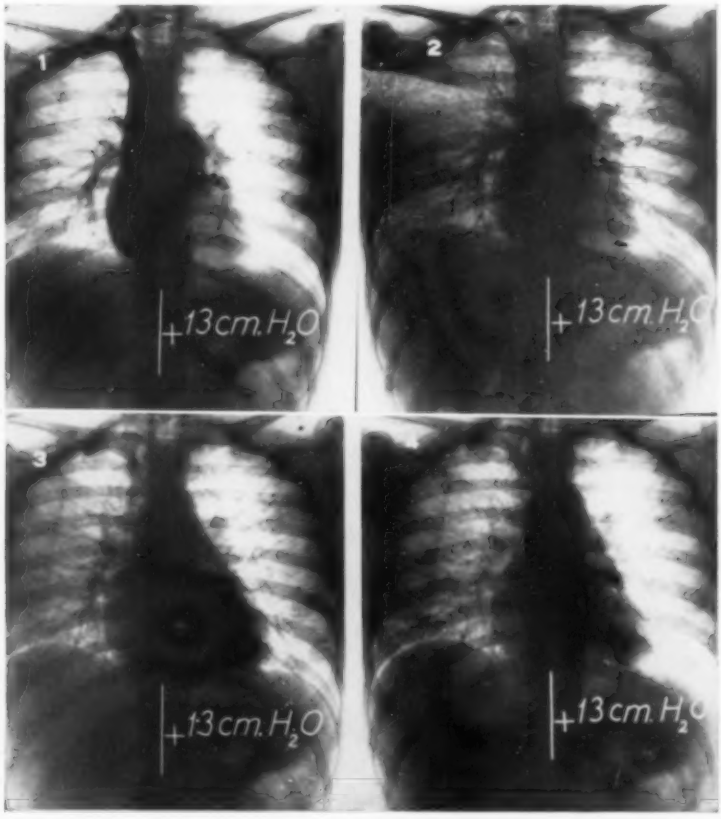

FIGURE 2: 23 year old woman, normal; see text.

(which undergo the positive pressure) do not appear in film 1, are evident in film 2 and remain without any further great change up to film 4, when the opaque substance has practically abandoned the unexpanded pulmonary zones (left lung and right superior lobe).

Figure 4 shows four films obtained in a woman 22 years old suffering from a right constrictive pachypleuritis subsequent to a pneumothorax and a small cavity of the right apex. A positive pressure of 25 cm. of water was maintained (in the left main bronchus) with nitrogen. The shift of the mediastinum to the right, the lowering of left leaf of the diaphragm and the increased radiolucency of the left half of the thorax existed before the test. The opaque substance circulates mostly through the sick lung as it is shown by the opacity of the right half of the thorax that attains its maximum in film 2 and decreases in the following. There are signs of circulatory delay in the left lung, in which the pulmonary artery remains unchanged in the first three films of the series.

The results obtained can be compared in the 10 cases we studied, taking,

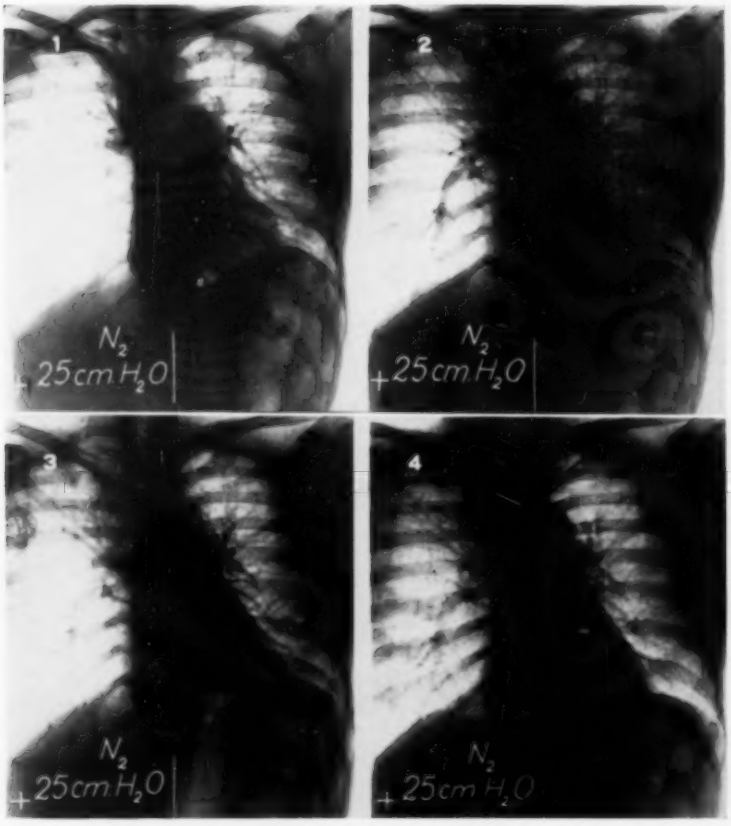

FIGURE 3: 38 year old woman, normal; see text.

of course, into consideration the anatomopathological differences between the areas through which we tried to deviate the blood flow and the different pressures used (between +13 and +25 cm. of water). These results demonstrate the efficiency of the suggested method as a means of blood shunting; in fact, the angiopneumograms show that in the territories which undergo the positive pressure, the contrast substance does not appear clearly, or is delayed, or is stagnant longer than in normal areas. These serious circulatory alterations, of the chronclogical type, correspond evidently to a great decrease in blood flow. These tests also confirm the hypothesis that the blood flow through a certain territory can naturally and easily increase with corresponding improvement of the respiratory function when the hydraulic resistance of the remaining circuits increases.[14, 16, 17]

From the theoretical point of view, it is important to notice that the increase of the alveolar pressure acts in two different ways: 1) it com-

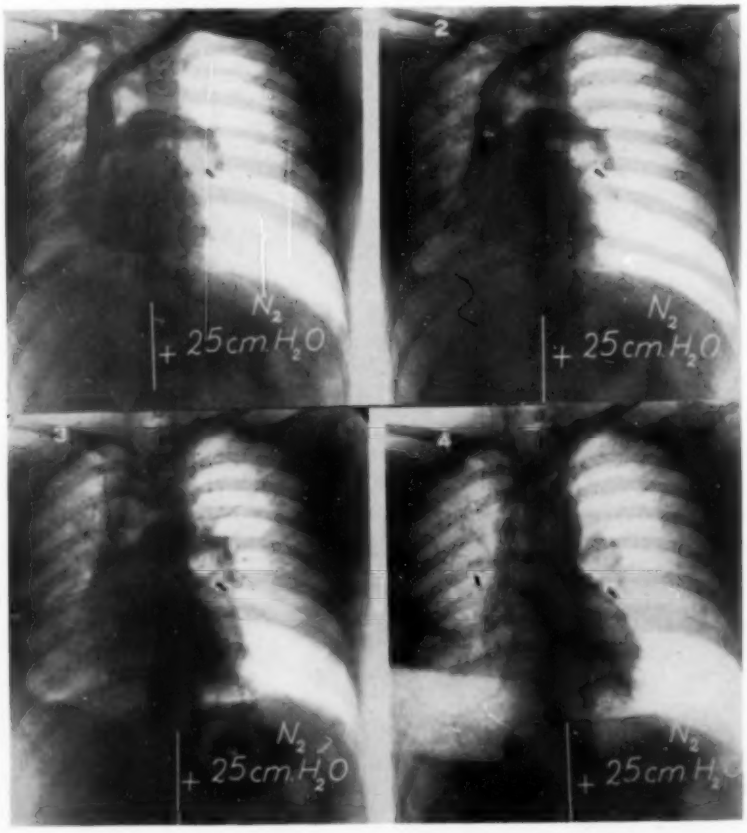

FIGURE 4: 22 year old woman, with right constrictive pachypleuritis, subsequent to a pneumothorax; see text.

presses the collapsed pulmonary veins and therefore reduces the circulatory gradient in the territory under positive pressure; 2) it reduces the vascular bed and therefore increases its resistance. It is due to this double effect that relatively small increases in pressure cause relatively marked results, even if the complete circulatory blockade can only be achieved when the intraalveolar pressure is greater than the maximum pulmonary arterial pressure.

For this reason, the present method might be in a way inferior to that due to the Swedish authors,[6] particularly in cases in which the pressure of the pulmonary artery is high, even if the field it covers is similar. In fact the blood deviation obtained with the unilateral endobronchial positive pressure is sufficient to eliminate a great part of the blood flow from the distended pulmonary areas; it can consequently give information on the *potential functional value* of the remaining parenchyma and could help in the clinical valuation of the cardiac reaction to a quick increase of the circulatory resistance in a pulmonary territory. The technique herewith described reduces the ventilation and the mobility of the half of the thorax which undergoes the positive pressure while this does not happen with the temporary occlusion of a pulmonary artery.

The increase of pressure does not cause dangerous action on the pulmonary parenchyma. Much greater increases take place with the Valsalva test during which the pulmonary pressure can reach a value six times higher than the maximum pressure we used. The only limitation might be the displacement of the mediastinum which, however, has been minimal and well tolerated with the pressures we used. Lastly, it should be remembered that this technique is a simple and easy means for a blood flow shunt within the possibilities of any hospital.

We come now to some deductions from the examination of the bronchospirometric tracings. Figure 5 represents the same normal woman as in figure 3. The spirometric tracings of both lungs are similar. The 20 cm. of water positive pressure applied with nitrogen to the right lung caused an oxygen absorption in the left lung in an amount approximately the sum of the consumptions of both lungs before the test. The same

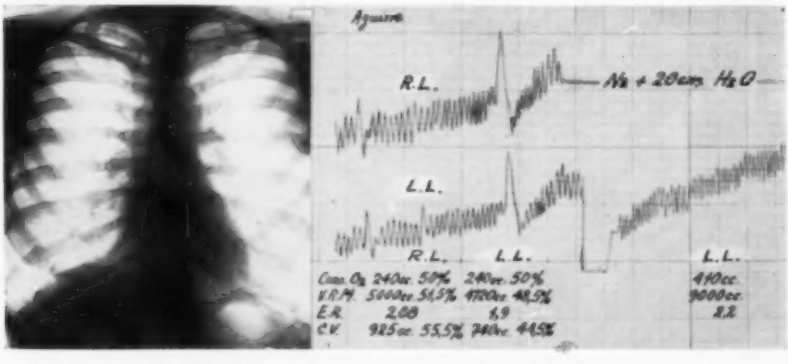

FIGURE 5: 37 year old woman, normal; see text.

thing happens with the ventilation while the respiratory equivalent does not change.

Figure 6 represents a woman 16 years old with residual fibrous lesions in the right lung. The minute oxygen absorption of this lung was 110 cm.^{3} before the test. The 20 cm. of water positive pressure applied with nitrogen to the healthy left lung increased the minute oxygen absorption of the sick lung up to 350 cm.^{3} This is nearly the sum of the two consumptions before the test. The ventilation of the sick lung also increases, but in a smaller proportion than the oxygen uptake, so that an increase of the respiratory equivalent (from 5.8 to 2.8) of this lung takes place.

Figure 7 represents a 22 year old woman with a right constrictive pachypleuritis subsequent to pneumothorax (the same as in figure 4). The minute oxygen absorption of the sick right lung was of 30 cm.^{3} before the test. The 20 cm. of positive pressure with water applied with nitrogen to the healthy left lung increased the oxygen absorption of the sick lung up to 285 cm.^{3} The ventilation also increased, but in a much smaller pro-

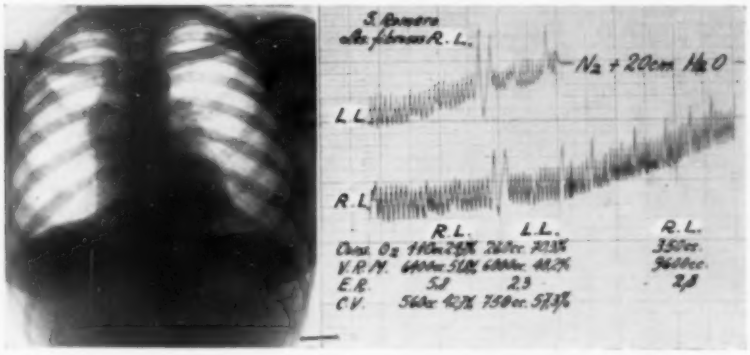

FIGURE 6: 16 year old woman, with residual fibrous lesions in the right lung; see text.

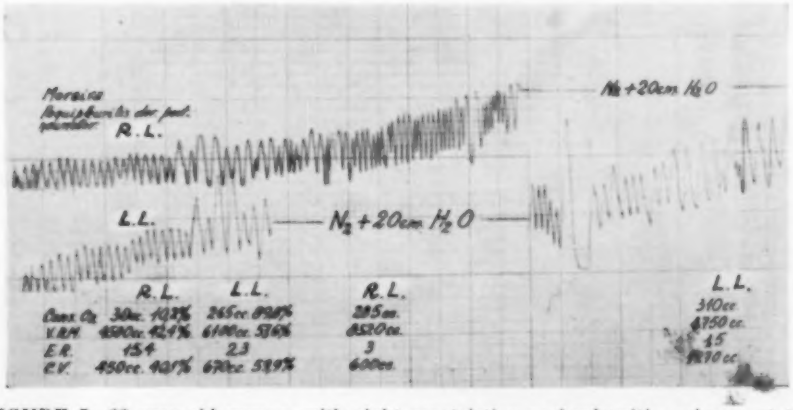

FIGURE 7: 22 year old woman, with right constrictive pachypleuritis, subsequent to a pneumothorax; see text.

portion, so that an almost complete correction of the respiratory equivalent took place (from 15.4 to 3). The positive pressure in the healthy lung was tolerated well, showing that a seriously sick lung, which previously carried only 10 per cent of the respiratory function, can handle the total amount when circumstances make it necessary at least while the patient is at rest. The positive pressure in the sick lung shows that the spirometric tracing of the healthy lung has characteristics similar to those of the tracing obtained before the test.

Figure 8 represents a man 25 years old with a right retroclavicular cavity who had to have a thoracoplasty. When the sick lung underwent 20 cm. of water positive pressure with nitrogen, oxygen absorption in the opposite lung increased from 170 to 295 cm.[3] The total absorption of both lungs before the test was 270 cm.[3] When the sick lung ventilates nitrogen at atmospheric pressure, the oxygen absorption of the left lung increases up to 450 cm.[3]

The results of all these bronchospirometric tests permit the deduction that, during unilateral intrapulmonic positive pressure, the absorption of the lung that breathes oxygen normally has a value close to the one corresponding to the total uptake of both lungs before the test. This observation is in accordance with the hypothesis that during unilateral positive pressure an important deviation in the blood flow towards the normally breathing lung occurs. The ventilation of the latter increases in a smaller proportion than the oxygen uptake with the corresponding improvement of the respiratory equivalent. Phenomena practically equivalent to the above mentioned appear when the unilateral intrapulmonic positive pressure is produced with oxygen instead of nitrogen.

We have regularly observed that the excessive absorption of oxygen in one lung, when the opposite ventilates nitrogen at atmospheric pressure, is greatly reduced when the pressure in the latter increases. This phenomenon is explained by the fact that the ventilation of a lung with nitrogen causes a "washing" of the oxygen from the alveoli and from the

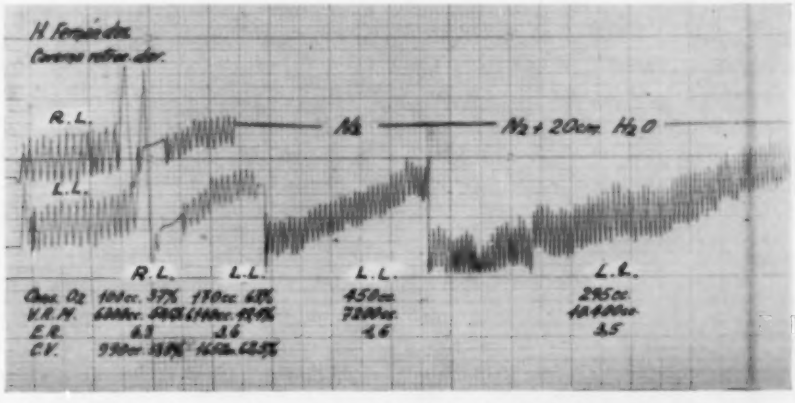

FIGURE 8: 26 year old woman, with right retroclavicular cavity; see text.

blood, and an equivalent amount of oxygen must be absorbed by the oxygen breathing lung. This loss of oxygen is greatly reduced by the positive pressure, and this is also in accordance with the hypothesis that the mentioned positive pressure causes an important reduction of the circulatory flow in the compressed lung.

The importance of the bronchospirometric tracings, from the point of view of a quantitative determination of the blood flow deviation, is reduced due to the fact that the positive pressure can cause hypoventilation in the compressed lung, which could itself force increased oxygen uptake in the opposite lung without the corresponding shunt of the blood flow.* We ignore at what point our experimental conditions may increase the mechanical work of breathing and consequently the oxygen requirements of the patient.[10]

SUMMARY

A description is given of a method of blood shunt from a particular lung area towards the remaining pulmonary parenchyma, through local hypertension obtained by connecting the bronchus of the area under study with an atmosphere of positive pressure of 25 cm. of water or less.

The blood flow reduction, in the areas which undergo positive pressure, was demonstrated by means of angiopneumography on 10 patients: four normal, and six with unilateral pulmonary tuberculosis. Therefore, this technique permits estimate of the *potential functional value* of the remaining parenchyma.

As a complementary work, on a total of 40 subjects (6 normal and 34 patients with unilateral pulmonary tuberculosis), we have studied the alterations in the spirometric tracing of an oxygen breathing lung at atmospheric pressure, when the other lung was ventilated in open circuit with nitrogen at atmospheric or positive pressure or with oxygen at positive pressure.

RESUMEN

Se pasan brevemente en revista los métodos descritos hasta el momento para el estudio de la función de los dos pulmones por separado.

Se describe un método de derivación sanguínea desde un área pulmonar determinada hacia el restante parénquima pulmonar, por medio de la hiperpresión localizada obtenida al conectar el bronquio del territorio estudiado con una atmósfera de presión positiva (de hasta +25 cm. de agua).

La disminución del flujo sanguíneo, en los territorios sometidos a la presión positiva, ha sido demonstrada por medio de la angioneumografía en 10 pacientes: 4 normales y 6 con tuberculosis pulmonar unilateral. Esta

*We have tried to complement the bronchospirometric experiments with oxymetric tests, that would have enabled us to evaluate the pulmonary blood flow variations. In fact the lowered arterial oxygen saturation (due to the blood shunt and to the oxygen ejection), which takes place during the unilateral ventilation with nitrogen at atmospheric pressure, would have had to disappear or greatly decrease during the unilateral positive pressure breathing, the effects of which are exactly the elimination of the blood flow from pulmonary compressed sections. We have used, with this aim, Milikan's anoxia photometer, model 17-410, manufactured by Coleman Instr. Inc., but a serious defect in the instrument, which could not be repaired in our country, made it impossible to use this test.

técnica da así la posibilidad de apreciar el *valor funcional potencial del parénquima restante.*

Se discuten las ventajas y los inconvenientes del método descrito.

Como investigación complementaria, en un total de 40 pacientes (6 normales y 34 con tuberculosis pulmonar unilateral), se ha buscado de estudiar las modificaciones que se producen en el trazado broncoespirométrico de un pulmón que respira oxígeno a presión atmosférica, cuando el otro pulmón es ventilado en circuito abierto con nitrógeno a presión atmosférica o a presión positiva, o con oxígeno a presión positiva.

RESUME

Les auteurs donnent la description d'une méthode qui permet de créer un shunt sanguin à partir d'une zone pulmonaire particulière vers le reste du parenchyme pulmonaire. Il est obtenu grâce à une hypertension locale due à la connection de la bronche à la zone en observation sous pression positive de 25 cm. d'eau au moins.

La réduction du débit sanguin dans les zones sous pression positive fut démontrée au moyen de l'angiopneumographie chez 10 malades: quatre sains, et six atteints de tuberculose pulmonaire unilatérale. C'est pourquoi ce procédé permet d'estimer la valeur fonctionnelle potentielle du reste du parenchyme.

Comme étude complémentaire, sur un total de 40 sujets (6 normaux et 34 atteints de tuberculose pulmonaire unilatérale) les auteurs ont étudié les modifications du tracé spirométrique de la respiration pulmonaire oxygénée à la pression atmosphérique normale alors que l'autre poumon était ventilé en circuit ouvert avec azote à la pression atmosphérique ou en pression positive ou avec oxygène en pression positive.

ZUSAMMENFASSUNG

Es wird eine Beschreibung gegeben über eine Methode eines Blut-Shunts eines bestimmten Lungenbezirkes dem übrigen Lungengewebe gegenüber, und zwar durch eine örtliche Hypertension auf dem Wege über die Verbindung des Bronchus des zu untersuchenden Bezirkes mit einer Atmosphäre eines positiven Druckes von 25 cm Wasser oder weniger.

Die Verminderung der Durchblutung in den Bezirken, die einem positiven Druck unterliegen, wurde nachgewiesen mittels Angiopneumographie an 10 Patienten: 4 normalen und 6 mit einseitiger Lungentuberkulose. Daher erlaubt diese Technik, den potentiellen Funktionswert des übrigen Parenchyms abzugrenzen.

Als ergänzende Arbeit haben wir an insgesamt 40 Personen (6 normalen und 34 mit einseitiger Lungentuberkulose) die Veränderungen in den spirometrischen Aufzeichnungen untersucht an einer mit Sauerstoff beatmeten Lunge unter atmosphärischem Druck, wenn die andere Lunge in einem offenen Kreislauf ventiliert wurde mit Stickstoff unter atmosphärischem oder positivem Druck oder mit Sauerstoff unter positivem Druck.

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Resuscitation from Cardiac Arrest Due to Acute Coronary Thrombosis*

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Introduction

Death from coronary heart disease may be due to a disorganization of rhythm resulting in arrest more often than it is due to actual failure or to rupture of the myocardium.[4,5] This probably means that some cases of death from coronary thrombosis could be saved if cardiac massage were available at the time of the fatal heart attack. Lending support to this idea are two recent reports of cardiac arrests secondary to myocardial infarction with successful resuscitation by Beck[2] and Regan[8]. This article reports a similar case in which a hospital employee stricken in the hospital corridor was resuscitated from a cardiac arrest secondary to myocardial infarction.

Mr. C. S., an Ancker Hospital orderly, was found unconscious in the main hallway of the hospital on March 19, 1956, at 4:53 P.M. Although his fall was not witnessed, he was discovered almost immediately because of heavy traffic in the hall. First examination revealed no respirations and no heart sounds. He was given artificial respiration and immediately taken to the receiving room where oxygen was administered by mask. Again no heart sounds were audible so 1 cc. of 1:1000 epinephrine was injected directly into the left ventricle but there were still no heart tones. The chest was then opened through a left thoracotomy incision in the third interspace. The pericardium was incised and the heart observed to be in standstill. Cardiac massage was begun at a time estimated to be three and one-half minutes after arrest occurred.

After a few minutes of massage, ventricular fibrillation set in so molar sodium lactate was injected into the left ventricle[3] and electric defibrillation carried out.[6] Regular pulsations began promptly and persisted. Spontaneous respirations started as soon as cardiac massage was initiated.

After closing the chest, he was taken to the post-operative ward, but it was necessary to reopen the wound one hour later because of a bleeding vessel in the wound.

Forty-eight hours later he responded to questions and was able to talk coherently. He became oriented and it was felt that he suffered little, if any, brain damage. But in spite of intensive antibiotic therapy, his wound became infected. Chest x-ray films were essentially clear until March 31, 1956, when evidence of pneumonia was seen. Tracheotomy and secondary closure of the infected wound were carried out on March 31, but on April 1 he expired.

Electrocardiogram on March 24, the sixth day after the attack, revealed an S-T elevation in Lead 1 and in the right precordial leads. This was interpreted as suggesting a possible recent anterior infarction. (Leads 1 and V1, V2, V3, V4.)

Post mortem examination revealed bilateral bronchopneumonia, atelectasis, pulmonary congestion and edema. The coronary arteries showed areas of occlusion in the left circumflex branch and the posterior descending branch of the right coronary. There was an area of hyperemic mottling in the base of the anterior portion of the interventricular septum.

Discussion

The technique of cardiac massage and defibrillation has been described many times[1, 4, 6, 7]. On the cases we have done, the injection of adrenalin has been helpful. We have also used several cc's of calcium gluconate followed by ½ to 1 cc. 1:1000 adrenalin. This usually seems to make the fibrillation active and consequently facilitate defibrillation.

*From the Department of Surgery, Ancker Hospital, St. Paul, and the University Hospitals, Minneapolis.

The fact of this temporary survival and the reported permanent survivals[2, 8] adds weight to the suggestion that a few cases of cardiac arrest secondary to coronary thrombosis can be saved. Cases which occur in the hospital, of course, are probably the ones that will be found early enough to resuscitate. In our patient the thoracotomy was done by an intern. His quick thinking and aggressive attitude made the resuscitation possible. Since this time, three other attempts have been made by the intern-resident staff. It would seem to be worthwhile to prepare interns and residents for this emergency if success is to be achieved. We have now set up sterile cardiac arrest packs with the necessary equipment and have one on each of the surgical floors and in the receiving room. Doubtless, these packs should also be kept on each of the medical floors where coronary attacks are more likely to occur.

Although two case reports with resuscitation from otherwise fatal coronary thromboses have appeared, failures may occur even when cardiac massage is instituted quickly. The case we report represents one in which a wound infection probably played a major role in the patient's death. Recently we had another case of cardiac arrest which was resuscitated successfully only to die two hours later of a ruptured myocardium in the area of infarction.

CONCLUSION

1. A case of cardiac arrest due to myocardial infarction in a hospital employee with successful resuscitation and survival for two weeks is presented.

2. Some cases of cardiac arrest secondary to an infarct probably can be resuscitated since death in these cases is often due to a disturbance of rhythm rather than myocardial failure.

3. An active interest in therapy for cardiac arrest in a resident-intern group of any hospital should produce, in time, some salvage from cardiac arrest secondary to coronary thrombosis.

CONCLUSION

1. Se relata el caso de un empleado de hospital que sufrió paro cardiaco debido a infarto del miocardio en el que se llevaron a cabo las manobras llamadas de "resucitación" con sobrevida por dos semanas.

2. Algunos casos de paro cardiaco secundario a infarto probablemente pueden rescatarse puesto que la muerte en estos casos se debe más bien a un trastorno del ritmo más que a desfallecimiento del miocardio.

3. Si se pone intrés en el tratamiento del paro cardiaco en un grupo de internos-residentes de cualquier hospital, con el tiempo se obtendrá el rescate de algunos casos de paro consecutivo a trombosis coronaria.

RESUME

1. Les auteurs présentent un cas d'arrêt cardiaque dû à un infarctus du cyocarde chez un employé de l'Hôpital, avec résurrection et survie de deux semaines.

2. Quelques malades ayant subi un arrêt cardiaque secondaire à un infarctus peuvent être réanimés puisque la mort apparente dans ces cas est souvent liée à une modification du rythme plutôt qu'à une insuffisance myocardique.

3. Un groupe de médecins résidant dans chaque hôpital devrait être spécialisé dans le traitement de l'arrêt cardiaque pour permettre une intervention suffisamment précoce et une résurrection dans les arrêts cardiaques par thrombose coronarienne.

SCHLUSSFOLGERUNGEN

1. Vorstellung eines Falles von Herzstillstand infolge Herzmuskel-Infarkt bei einem Angestellten eines Krankenhauses mit erfolgreicher Wiederbelebung und Weiterleben von 2 Wochen.

2. Gewisse Fälle von Herzstillstand im Gefolge eines Infarktes können wahrscheinlich ins Leben wieder zurückgerufen werden, weil der Tod in diesen Fällen öfter die Folge einer Rythmusstorung ist als eines Versagens des Herzmuskels.

3. Ein aktives Intersse an der Therapie des Herzstillstandes in einer Gruppe von internistischen Assistenten eines Krankenhauses müsste auch gelegentliche Rettung bein Herzstillstand im Gefolge von Coronar-Thrombose bewirken.

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The Management of Tuberculous Empyema*

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Tuberculous empyema is usually caused by rupture of a subpleural parenchymal focus through the visceral pleura. The immediate result of such a rupture is the occurrence of hydrothorax, the so-called idiopathic pleural effusion. In most instances, particularly with adequate drug treatment, the site of rupture seals. The fluid is then absorbed or removed by thoracentesis and the lung re-expanded, usually with little residual evidence of parenchymal disease. Too often, however, the bronchopleural fistula remains open and the hydrothorax converts into pyothorax, either pure tuberculous or mixed. The scene is then set for the tragic course which in 1915 caused Robinson to designate tuberculous empyema as the "Patriarch of the Surgical Scrap Heap."

The occasional demonstration of acid-fast organisms in the bone marrow, or of miliary tubercles in the liver, of patients with tuberculous empyema has been cited by some as evidence that the initial pleural effusion is only one phase of a tuberculous bacteremia which results from a lympho-hematogenous progression of a primary pulmonary focus.

These bone marrow and liver signs may be explained on a purely arithmetical basis. As long as the lesion is confined to the pulmonary parenchyma the lymphatic drainage is restricted to the hilar nodes and the chance of progression to the thoracic duct and the systemic circulation is small (Fig. 1). When, however, the lesion ruptures into the pleural space the possibility of lympho-hematogenous extension is increased (Fig. 2). In addition to the hilar nodes, the pleural lymphatics also drain through the paravertebral, the paraaortic and parasternal lymph plexuses. There are thus four times as many opportunities for pleural infection to reach the systemic circulation as when the infection remains restricted to the parenchyma.[1] It is not surprising, therefore, that one is occasionally able to find lesions in the bone marrow, spleen, liver or kidneys in patients with tuberculous empyema.

Auerbach's[2] demonstration of a bronchopleural fistula in 49 per cent of 311 autopsies done in patients who died with tuberculous empyema would seem to be conclusive evidence as to its pathogenesis.

Once empyema is established, a serious threat to the patient's life is presented. We are all too familiar with the chain of events; sepsis, amyloidosis, progressive tuberculous pulmonary fibrosis and insufficiency, which exacted such a toll in pre-chemotherapy days. Physicians often differed as to the best method of treatment of this serious condition but there was little difference of opinion as to the high morbidity and mortality rates that were encountered.

The mortality rate for pure tuberculous empyema ranged from 70 to

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90 per cent when treated only with thoracentesis and irrigation. If thoracoplasty was done, the mortality rate of 15 to 40 per cent was still forbidable. In patients with mixed empyema the rates were even higher.

The few who recovered after numerous stages of thoracoplasty and repeated chest wall excisions were left with such a skeletal deformity and reduced pulmonary function that life held few remaining compensations.

When streptomycin was introduced in 1946, efforts were directed toward reducing the hazards of empyema through its use. The Veterans Administration-Armed Forces Cooperative Conference drew up protocols under which alternate cases of tuberculous empyema were treated either with parenteral and intrapleural streptomycin, and irrigation, or with simple irrigations without streptomycin. The failure of streptomycin to significantly reduce mortality rates was often attributed to the fact that the Ph of the pleural fluid was too low or too high, according to one's opinion as to the acidity or alkalinity of the pleural exudate.

After two years of effort, the Conference realized that pyrothorax was usually the end result of an uncontrolled pulmonary lesion and not a disease entity. Treatment directed only toward sterilizing the pleural cavity, therefore, could only result in failure. The empyema protocols were then abandoned and efforts directed toward control of the pulmonary lesions and a more definite surgical approach when faced with an established empyema.

This method of attack has resulted in marked decrease in the incidence of empyema just as it has in all forms of extrapulmonary tuberculosis. When, however, empyema does develop management of the patient must be carefully planned if we are to prevent the unhappy sequence of events described above.

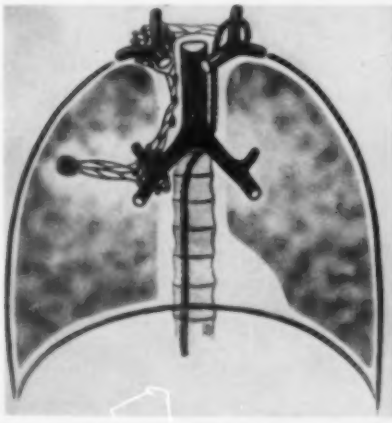

FIGURE 1

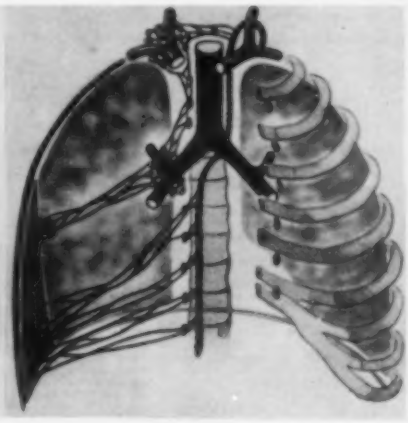

FIGURE 2

Figure 1: Lymphatic Drainage when Tuberculous Lesion is Restricted to the Pulmonary Parenchyma (Courtesy W. W. Stead, M.D.).—*Figure 2:* Avenue of Lymphatic Drainage when Parenchymal Lesion Ruptures into the Pleural Space (Courtesy W. W. Stead, M.D.).

The best treatment is prophylactic. Early diagnosis of pulmonary tuberculosis followed by prolonged, continuous treatment with one of the effective drug regimens will continue to result in a decreasing incidence of pleural infections. The incidence of empyema resulting from a complication of pulmonary resection can be further reduced by adequate pre- and postoperative chemotherapy and timing of the operation so that the maximum degree of stability of the parenchymal lesion will have been attained prior to surgery. When it becomes necessary to resect in the presence of an open cavity and positive sputum, the complications are definitely increased. It is not necessary to emphasize here the necessity for painstaking surgical technique and alert pre- and postoperative supervision. Of paramount importance is the early elimination of any postresection intrapleural air space.

When empyema has developed the situation is emergent and active measures are required if catastrophe is to be prevented. There are several objectives which must be satisfied if success is to crown our efforts. Among these are; elimination of toxicity, stabilization of the disease process and obliteration of the empyema space. The attainment of these objectives may in turn make possible, or require excision of the damaged lung tissue, or surgical collapse measures when excision is not feasible.

The sequence in which these objectives are approached must be varied to meet the existing situation. If the empyema is acute and is associated with an exudative parenchymal lesion in a febrile toxic patient, the defense mechanisms must be marshaled in an orderly and precise manner. Our practice is to place the patient on one of the several satisfactory chemotherapy regimens immediately and to do closed thoracotomy. A No. 20 French or larger rubber catheter is inserted in an appropriate interspace and suction equivalent to a negative pressure of 10 to 15 cm. of water applied. It has been our experience that such an amount of suction improves drainage and frequently encourages re-expansion without reopening a bronchopleural fistula. Needle aspiration is not done after the first diagnostic puncture because of the tendency to develop needle tract abscesses.

Drug treatment must be continued for an indefinite period, at least one year and commonly two years. After two months of treatment, or earlier if conditions warrant, a reappraisal is made. If the course is to be benign, obliteration of the empyema space will have been accomplished by re-expansion of the lung and elimination of the exudate. Treatment with drugs can then be continued and necessary surgical measures introduced at a suitable time as in any case of parenchymal disease without empyema.

If, however, the space does not obliterate after six-to-eight weeks of treatment, more active measures are demanded. In such patients, particularly if the disease is confined to one lung, surgical intervention is required at a much earlier time than in patients under drug therapy for acute parenchymal disease. If the objective of removal of toxicity is to be satisfied, this empyema space must be obliterated. It is our practice under such conditions to do an extensive thoracoplasty before proceeding

to resection. Occasionally by doing a prior thoracoplasty the need for resection may be removed. If resection proves necessary it can then be done on a patient in better condition and with less postoperative disturbance than if resection is done at the height of the inflammatory process.

A more chronic type of empyema is sometimes encountered. This is one in which the empyema represents a complication of some type of treatment, often, in former years, pneumothorax or pneumonolysis done in the attempt to improve the collapse. Under such circumstances there is more time available to plan the treatment course and to marshal the patient's reserves. The empyema has not developed in virgin soil since a defensive barrier of fibrosis had already been erected as a result of the treatment program. Chemotherapy is initiated or continued as the situation demands. Deficiencies in blood volume are corrected and electrolyte supplied if necessary. Avitaminosis is remedied. If the parenchymal lesion is stable and resection not necessary, the sputum negative by culture, and bronchopleural fistula is not present, it is usually possible to proceed with decortication depending upon rapid re-expansion of the lung to eliminate the empyema pocket.

There remains, however, a third group of patients who fall midway between the acutely ill and the chronic type. They have a considerable amount of parenchymal disease, frequently bilateral in distribution and unstable in character. A cavity may be present and bronchopleural fistula is present or has been demonstrated at some time during the course of the empyema. These patients are chronically ill, have a depletion of their blood volume, an electrolyte imbalance and frequently amyloidosis. Fortunately, with the modern drug treatment of tuberculosis, they are being encountered with decreasing frequency. Each step of the treatment program must be carefully planned. The blood volume and depleted protein reserves are restored by means of blood transfusions. Vitamin deficiencies and electrolyte imbalances are corrected. At the initial thoracentesis we insert a Pezzer catheter and attach a suction pump set at 10 to 15 cc. of water negative pressure. We commonly use varidase or tryptar instillations after drainage is established, not with much hope of eliminating the thickened membrane but with the objective of cleaning up the empyema pocket prior to the necessary reparative surgery.

Opinions differ as to the best corrective measure to apply in such circumstances. Many surgeons proceed with thoracoplasty or with extrapleural pneumonectomy without preliminary drainage. In our minds the preliminary drainage serves not only to clean up the area prior to surgery but minimizes the possibility of spread of the purulent material to the contralateral lung while under general anesthesia.

Having initiated drainage and placed the patient in better clinical condition, we must not delay too long before instituting reparative measures. It is more dramatic to remove the pleural sac and the involved area of lung at the initial operation. In our hands, however, the complications following such procedures have been higher than in patients upon whom we did one or two stages of thoracoplasty followed by decor-

tication and resection of the lung or lobe as necessary. If the chest wall is collapsed prior to excision, the secondary infection which commonly develops is not nearly as difficult to handle as it is when the parietal pleura has been decorticated and the chest wall otherwise left intact. It is frequently necessary to do several subsequent modified Schede type of procedures unroofing and saucerizing sinus tracts before final success is attained.

DISCUSSION AND SUMMARY

In 1931 a Committee Report on the Treatment of Tuberculous Empyema[3] was submitted by Leroy S. Peters, Paul Ringer, J. J. Singer, and E. S. Welles. Among other conclusions were these:

1. Most tuberculous empyemata are best treated by no treatment.

2. Aspiration is justified to relieve high fever and pressure symptoms.

3. Closed or open drainage is not warranted since it converts a simple empyema into a pyogenic one.

In the years following that report and before the advent of successful chemotherapy, considerable reduction in mortality had been brought about by the more frequent use of thoracoplasty with or without preliminary drainage.

The demonstration of the bacteriostatic effect of streptomycin in 1946 opened the door for further improvements in the management of tuberculous empyema.

Active treatment has replaced inactivity. As soon as a diagnosis is established a carefully formulated program is set in motion with the objectives of elimination of toxicity, stabilization of the disease process and obliteration of the empyema space. These goals may occasionally be accomplished with chemotherapy and thoracentesis. Most commonly, however, chemotherapy accompanied by drainage, decortication, thoracoplasty, or resection is required.

DISCUSION Y RESUMEN

En 1931 se presentó un informe de un comité para el estudio del Tratamiento de Empiema Tuberculoso, por Leroy S. Peters, Paul-Ringer, J. J. Singer y E. S. Welles.

Entre otras conclusiones se encontraban las siguientes:

1. La mayoría de los empiemas se tratan mejor por la abstención.

2. La aspiración se justifica para aliviar la temperatura elevada y los síntomas de compresión.

3. La canalización abierta o cerrada no están justificadas puesto que convierten un empiema simple en un piogénico.

En los años siguientes a este informe y antes del advenimiento de la quimioterapia satisfactoria, se presenció una considerable reducción de la mortalidad por el uso más frecuente de la toracoplastía con o sin canalización preliminar.

La demonstración del poder bacteriostático de la estreptomicina en 1946 abrió la puerta para mayores mejoras en el tratamiento del empiema tuberculoso.

El tratamiento activo ha substituído al expectante. Tan pronto como el diagnóstico se establece se pone en práctica un plan cuidadoso para eliminar la toxicidad, para estabilizar el proceso patológico y la obliteración del espacio empiemático. Estos objetivos pueden ocasionalmente alcanzarse con la quimioterapia y la toracentesis. Más a menudo sin embargo, se requieren la quimioterapia acompañada de canalización, decorticación, toracoplastía o resección.

DISCUSSION ET RESUME

En 1931, un rapport sur le traitement de la pleurésie purulente tuberculeuse fut présenté par Leroy S. Peters, Paul Ringers, J. J. Singer et E. S. Welles.

Parmi d'autres conclusions, on peut relever celles-ci:

1. Pour la plupart des épanchements tuberculeux, le meilleur traitement est de n'en faire aucun.

2. L'aspiration est justifiée pour soulager la fièvre et les symptômes de compression.

3. Le drainage, ouvert ou fermé, n'est pas recommandé puisqu'il transforme un épanchement simple en épanchement purulent.

Dans les années suivant cette communication, et avant l'apparition de la chimiothérapie bienfaitrice, on a obtenu une réduction considérable de la mortalité par l'usage fréquent de la thoracoplastie avec ou sans drainage préalable.

La démonstration de l'effet bactériostatique de la streptomycine en 1946 ouvrit la voie à des améliorations ultérieures dans le traitement de l'épanchement tuberculeux.

Un traitement actif a remplacé l'absence de traitement. Dès qu'un diagnostic est établi, un programme soigneusement élaboré est mis en oeuvre pour éliminer la toxicité, stabiliser le processus de la maladie et compler l'espace occupé par l'epanchement. Ces desseins peuvent être atteints dans certains cas par la chimiothérapie et la thoracenthèse. Plus souvent cependant, il faut associer la chimiothérapie au drainage, à la décortication, à la thoracoplastie ou à la résection.

BESPRECHUNG & ZUSAMMENFASSUNG

Im Jahre 1931 wurde ein Ausschussbericht über die Behandlung des tuberkulösen Empyems[3] vorgelegt von Leroy S. Peters, Paul Ringer, J. J. Singer und E. S. Welles. Unter anderem ergaben sich daraus die folgenden Schlüsse:

1. Die meisten tuberkulösen Empyeme werden am besten behandelt, wenn sie nicht behandelt werden.

2. Eine Punktion ist gerechtfertigt, um hohes Fieber und Druckerscheinungen zu beheben.

3. Geschlossene oder offene Drainage ist nicht vertretbar, weil sie aus einem einfachen Empyem ein eitriges macht.

In den auf diesen Bericht folgenden Jahren und vor dem Eintritt er-

folgreicher Chemo-Therapie wurde eine beträchtliche Verminderung der Mortalität erzielt mit Hilfe des häufigeren Gebrauches der Thorakoplastik mit oder ohne einleitende Drainage.

Der Nachweis der bakteriostatischen Wirkung des Streptomycins im Jahre 1946 eröffnete die Voraussetzungen für eine weitere Verbesserung in der Behandlung des tuberkulösen Empyems.

Die aktive Behandlung ist an Stelle der Inaktivität getreten. Sobald die Diagnose feststeht, wird ein sorgfältig formuliertes Programm in Gang gesetzt mit dem Endziel der Auschaltung der Toxizität, Stabilisierung des Krankheitsprozesses und Obliteration der Empyemhöhle. Diese Absicht wird man gelegentlich mit Chemotherapie und Thorakozentese verwirklichen können. Für gewöhnlich ist jedoch die Chemotherapie in Gemeinschaft mit Drainage, Dekortikation, Thorakoplastik oder Resektion erforderlich.

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Non-Tuberculous Cavitary Disease of the Lungs*

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Introduction

Cavitary disease of the lungs is so frequently of tuberculous etiology that its demonstration almost invariably suggests an initial diagnosis of pulmonary tuberculosis. However, a wide variety of other conditions may produce or simulate cavity-formation within the lungs. In some cases, of course, the non-tuberculous nature of the underlying pathology may be readily apparent but in others the masquerade may be difficult to penetrate. Since therapy varies greatly, depending upon the etiology, it is important to avoid the pitfall of treating as tuberculous a cavity which is the result of a different pathologic process. The purpose of this study is to re-emphasize the multiplicity of non-tuberculous conditions which may produce or simulate cavitary disease of the lungs on x-ray examination.

The essential feature of pulmonary cavitation is necrosis of lung parenchyma with evacuation[1] of the resultant detritus and the development of a surrounding zone of reaction which largely determines the characteristics of the residual structural defect or "cavity." The classical pulmonary cavity is that seen in tuberculosis and in the strict pathologic sense the application of this term to non-tuberculous diseases should perhaps be limited to instances which closely parallel the tuberculous process. In this paper, however, the term has been used broadly and includes any condition of the lungs which may either produce excavation of pulmonary parenchyma or resemble it on x-ray film. In addition, extra-pulmonary entities which may be confused with pulmonary cavitation roentgenologically are briefly considered.

Material

Hermann M. Biggs Memorial Hospital is a 250-bed institution serving a semi-urban and rural area which is chiefly agricultural but which in places is heavily industrialized. Although primarily concerned with the prevention, diagnosis, and treatment of pulmonary tuberculosis, the hospital occasionally encounters instances of non-tuberculous pathology as diagnostic problems. In an effort to determine the extent to which cavitation may occur in, or be simulated by, non-tuberculous conditions, a survey was made of all such cases studied at this institution in the 13-year period between the opening of the hospital in 1937 and December 1950, a total of

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145 patients. Due to the lack of representation of certain diseases, such as coccidioidomycosis, and the paucity of others, such as bronchiectasis and the acute pneumonias, it was not hoped to secure data of overall statistical significance. With the advantage of unusually extensive serial roentgenograms on almost all patients, and pathologic data on many, it was possible in the vast majority of cases to follow the course of the disease from the first examination to the time of cure or death.

Results

Great difficulty may exist in properly categorizing instances of pulmonary pathology which exhibit cavitation. Frequently the picture is complicated by superimposed infection and the role of the initial lesion may be obscured. As far as possible, the instances reported were classified according to the primary or initiating lesion and frequently this was confirmed by pathologic data. Any individual who presented evidence of incontrovertible rarefaction or frank cavitation on one or more examinations was classified as showing "cavity on x-ray." In some cases cavitation was detected only by the use of special technics such as tomography.

Table I presents an analysis of the results obtained in the present study. Of the 145 cases which were reviewed, 141 were instances of acquired non-tuberculous pulmonary pathology and in 98 (70 per cent) of these cavitation was noted roentgenologically at some time in the course of the disease. Three cases of lung cyst were encountered and all showed pseudocavitation on x-ray film. In one additional patient the presence of a diaphragmatic hernia simulated cavitary disease of the lung roentgenologically.

From the aspect of the actual pathology involved it should be pointed out that Medlar[2] believes the "cavitary" changes discerned roentgenologically in many instances of suppurative bronchopulmonary disease, including abscess and bronchiectasis, represent extreme dilatation of bronchi rather than true lung destruction. Nevertheless, because the x-ray film incidence of cavitation in various disease entities is of greater clinical significance than its absolute incidence, reference was not made to pathologic data in preparing Table I, except where indicated to corroborate a diagnosis. Although excavation of pulmonary parenchyma was observed on pathologic examination of surgical or autopsy material in several patients who had not presented roentgen evidence of cavity-formation, in a greater number the reverse was true, pathologic study failing to disclose cavity-formation which had been thought to be present unequivocally on x-ray film. This suggests the importance of temporal relationships in a study of pulmonary cavitation and emphasizes the possible transitory nature of many cavities. A significant additional factor in the roentgen demonstration of such lesions is the presence or absence of a bronchial communication at the time of examination. Furthermore, evidence has recently been adduced[3] that some lesions which appear definitely cavitary on x-ray film actually contain solid or liquid material and hence are not cavities in the accepted sense.

TABLE I
INCIDENCE OF PULMONARY CAVITATION AMONG DISEASES STUDIED IN THE PRESENT SERIES

Disease	No. of Cases	Cavity on X-ray
Suppurative bronchopneumonia	1	0
Chronic lung abscess		
Staph., Strep., Pneumococcus, H. infl.	66	56
Friedländer's bacillus	3	2
Fusospirochetal	8	8
	77	66
Bronchiectasis	1	1
Fungus infection		
Actinomycosis	2	2
Aspergillosis	1	1
Moniliasis	3	1
	6	4
Foreign body	8	4
Neoplasm, malignant, primary		
Undifferentiated carcinoma	15	9
Squamous cell carcinoma	9	6
Adenocarcinoma	2	1
	26	16
Neoplasm, malignant, secondary		
Adenocarcinoma	9	5
Teratocarcinoma	1	1
Malignant giant cell tumor	1	0
Lymphosarcoma	1	0
Synovioma	1	0
	13	6
Infarct	2	1
Sarcoid	7	0
TOTAL	141	98
		approximately 70 per cent

In addition, three cases of lung cyst all showed pseudo-cavitation on x-ray film and one case of diaphragmatic hernia suggested pulmonary cavitary disease roentgenologically.

Discussion

Although our overall x-ray film incidence of cavitation in cases of non-tuberculous pulmonary pathology (70 per cent) may appear inordinately high, it will be recalled that diagnostic criteria included "rarefaction" as well as the larger and more striking cavitary lesions. Since cavitation may be transient, its incidence may well increase in direct proportion to the frequency of x-ray film examinations. The results are further modified by the fact that the period of study included several years of the pre-antibiotic era when the advantages of modern specific therapy were not available. With these factors considered, it is felt that the present data convey a valid impression regarding the roentgenologic occurrence of

TABLE II—NON TUBERCULOUS PULMONARY CAVITATION

A. Entities which may be associated with definite cavitation both roentgenologically and pathologically.

I. Pneumonitis:
- a. Lobar pneumonia
- b. Primary atypical (virus) pneumonia
- c. Suppurative bronchopneumonia
- d. Brucellosis

II. Chronic lung abscess:
- a. Staph., Strep., Pneumococcus, H. infl., and other related bacteria
- b. Friedländer's bacillus
- c. Fusospirochetal

III. Bronchiectasis.

IV. Fungus and yeast infection:
- a. Coccidioidomycosis
- b. Actinomycosis, including nocardiosis
- c. Aspergillosis
- d. Blastomycosis
- e. Histoplasmosis
- f. Streptothricosis
- g. Geotrichosis
- h. Cryptococcosis (torulosis)
- i. *A. boydii* infection

V. Parasitic disease:
- a. Amebiasis
- b. Hydatidosis
- c. Paragonimiasis

VI. Foreign body.

VII. Neoplasm:
- a. Benign
  - 1. Bronchial adenoma
- b. Malignant
  - 1. Primary: carcinoma and sarcoma, including Hodgkin's disease and other lymphomas
  - 2. Secondary: carcinoma and sarcoma, including Hodgkin's disease and other lymphomas; myelogenous leukemia

VIII. Pulmonary infarction.

IX. Allergy and collagen diseases:
- a. Loeffler's syndrome
- b. Polyarteritis
- c. Systemic lupus erythematosus
- d. Wegener's granulomatosis

X. Pneumoconiosis:
- a. Silicosis
- b. Anthracosilicosis

XI. Sarcoidosis.

XII. Syphilis (gumma).

XIII. Aortic aneurysm.

XIV. Atelectasis.

XV. X-ray therapy.

B. Intra-pulmonary conditions which may appear to demonstrate cavitation roentgenologically but which are not characterized pathologically by necrosis:
- I. Pulmonary emphysema
- II. Congenital pulmonary cysts and allied cystic lesions, including those seen in xanthomatosis
- III. Pulmonary vascular shadows
- IV. Trauma to lung parenchyma

C. Extra-pulmonary entities roentgenologically simulating cavitary lung disease:
- I. Encapsulated empyema
- II. Loculated pneumothorax
- III. Diaphragmatic hernia
- IV. Uremia
- V. Calcification in first costal cartilage
- VI. Film artifact

D. Miscellaneous:
- I. Experimental intra-parenchymal injection of egg albumin[77]
- II. Post-resection pseudo-cavity[78]

cavitation in non-tuberculous pulmonary disease. However, because of limitations in clinical material, it was thought desirable to supplement these results with a review of the literature. Based on such a survey, Table II enumerates entities which have been found, constantly or inconstantly, to be associated with either true or apparent pulmonary cavitation.

Pneumonia: Even with present-day therapy, pneumonia is of relatively frequent occurrence among the non-tuberculous pulmonary diseases and cavitation may be associated with uncomplicated pneumonitis more commonly than is generally appreciated. Roentgen evidence of cavitation has been described in bacterial pneumonia of lobar distribution.[4, 5, 6] Since the course is usually benign and marked by spontaneous resolution, unnecessary anxiety may result from improper interpretation of the x-ray film. Rarefaction has also been noted during the resolution of primary atypical (virus) pneumonia.[5] Cavity-formation with fluid levels may be a conspicuous feature of acute suppurative bronchopneumonia,[7, 8, 9] particularly when seen in children. Brucellosis may uncommonly be associated with x-ray evidence of cavity, Greer[10] noting one instance in a series of 59 cases.

Lung Abscess: Because of the destructive nature of the pathologic process, chronic lung abscess might be expected to present a high incidence of excavation and in the present study this was found to be true. Seventy-seven patients with this condition were encountered and in 66 cavitation was demonstrable on x-ray film. Etiologically, various strains of *Staph-*

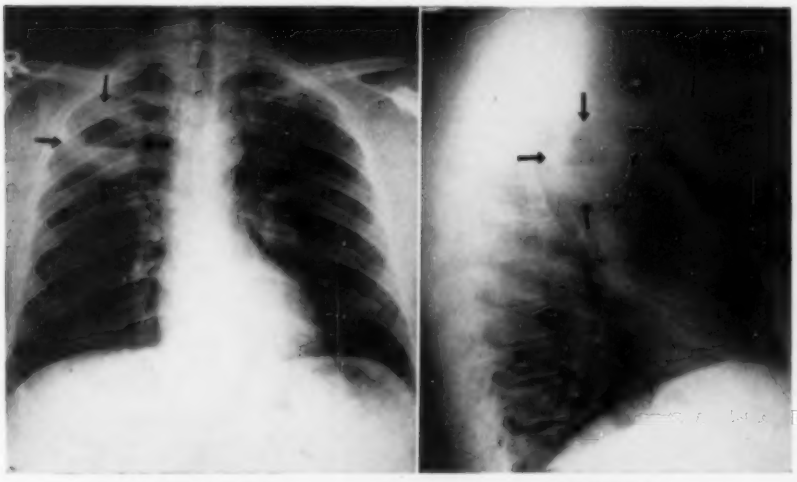

FIGURE 1A FIGURE 1B

Figure 1: Lung Abscess. Postero-anterior (a.) and right lateral (b.) roentgenograms demonstrating right upper lobe cavitation due to lung abscess. A fluid level is apparent. The patient was a 35 year old man with a two-month history of fever and productive cough. All sputum studies were negative for *M. tuberculosis*, but culture consistently yielded α-hemolytic *Streptococcus* and non-hemolytic *Streptococcus*. Specific antibiotic therapy was followed by prompt regression of symptoms and obliteration of the cavity. (Courtesy of Dr. John W. Strieder, Brookline, Mass.).

ylococcus, Streptococcus, Pneumococcus, and *Hemophilus influenzae* accounted for 66 cases, 56 of which showed cavity (Fig. 1). Three cases were due to Friedländer's bacillus and in two of these cavitary disease developed. All eight instances of fusospirochetal abscess were marked by cavitation.

Bronchiectasis: Pathologically, the association of cavitation with bronchiectasis has been described,[11] but in only a relatively small proportion of cases is it seen roentgenologically.[12] In the present series there was but one patient with uncomplicated bronchiectasis and this individual showed x-ray film evidence of cavity. Bronchography is, of course, indispensable in the diagnosis of bronchiectasis and may indicate the etiology of such cavities. (Fig. 2).

Fungus Infection: Certain pathogenic fungi and related organisms with or without a specific predilection for pulmonary parenchyma may produce cavitary lesions of the lungs. Most important among diseases of this etiology is coccidioidomycosis because it is of relatively common occurrence and frequently results in the formation of cavities which may resemble those due to tuberculosis. According to Smith *et al.*[13] the incidence of cavitation in inapparent coccidioidomycosis is not known but among a large number of patients hospitalized for this disease it ranged from 2 per cent to 8 per cent. Of the instances with cavitation studied by this group, 90 per cent of the cavities were single and 70 per cent were located in the

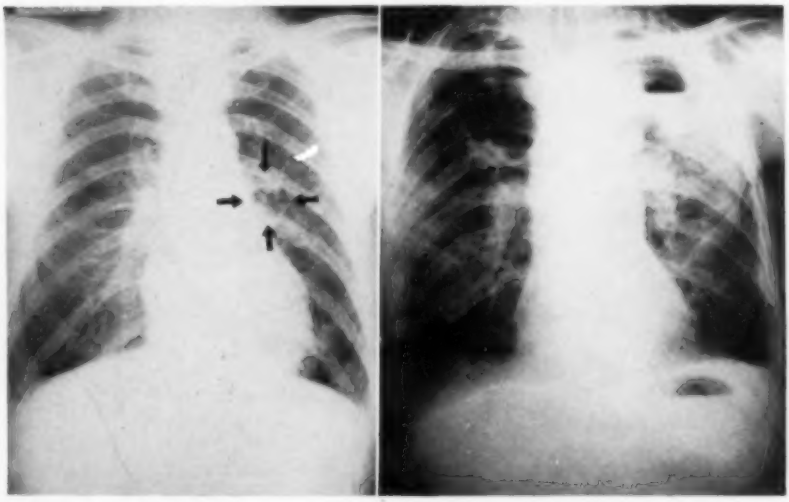

FIGURE 2 FIGURE 3

Figure 2: Bronchiectasis. Postero-anterior roentgenogram showing a thin-walled "cavity" associated with bronchiectasis which had been demonstrated bronchographically.—*Figure 3:* Aspergillosis. Left upper lobe cavitation in a 61 year old man whose bronchoscopic aspirate consistently yielded *Aspergillus fumigatus* on culture. No other pathogens were isolated despite intensive search. The lesion regressed after the institution of iodide therapy.

upper lung fields. In contrast to tuberculous cavities, those due to *Coccidioides immitis* are relatively benign and offer a more favorable prognosis. Although collapse therapy and excisional surgery have a definite role in the management of certain types of coccidioidal cavity,[14, 15] such measures are less commonly required than in similar cavities of tuberculous origin.[16]

Actinomycosis of the lungs is rather commonly associated with cavity-formation,[12] and two such cases were observed during the period of this study, both terminating fatally. Infection with *Nocardia asteroides*, a member of the family *Actinomycetaceae*, has been found to be associated roentgenologically and pathologically with cavitation and to mimic pulmonary tuberculosis in many respects.[17] Aspergillosis involving the lung is rare and when seen is usually due to *Aspergillus fumigatus*, although *A. niger* and *A. flavus* may less commonly be responsible. Pulmonary cavitation of this etiology has been noted by many[18, 19, 20, 21, 22] and one instance (Fig. 3) is included in the present report. According to Chortis[18] this disease is usually confused with pulmonary tuberculosis because it has a similar predilection for the upper lobes and apices. In a recent report[21] the case of a 51-year-old woman with post-influenzal bronchopneumonia is cited. Despite therapy with massive amounts of penicillin, streptomycin, and chloramphenicol the clinical condition rapidly deteriorated and x-ray films demonstrated a cavitating pneumonic process. At autopsy the left upper lobe was found to contain a cavity due to *A. fumigatus*. The authors conclude that the intensive antibiotic regimen may have permitted abnormal proliferation of fungi by suppressing bacteria which usually compete with the former for nutriment. Should this view prove correct, the present widespread use of antibiotics in pulmonary infections might witness an increase in the incidence of cavitary lesions due to fungi or related organisms.

No instance of pulmonary involvement with *Blastomyces dermatitidis* was seen in the present series. According to Martin and Smith[23] cavitary lesions due to pulmonary blastomycosis are uncommon and do not exceed 1 cm. in diameter, but in a more recent review Takaro and co-workers[24] found roentgen evidence of cavitation in six of 13 cases. Infection with *Histoplasma capsulatum* may account for many lung lesions formerly ascribed to *M. tuberculosis* and while cavitation may occur[25, 26] it is said to be extremely rare.[27] According to Golden[4] pulmonary streptothricosis may exist in a localized form and produce a cavitary lesion. Geotrichosis, while uncommonly involving the lung, may at times be associated with cavitation,[22] as also may be cryptococcosis (torulosis)[28, 29] and infection with *Allescheria boydii*.[30] Although usually considered as innocuous saprophyte, *Candida (Monilia) albicans* may exhibit pathogenic propensities when occurring in debilitated individuals. In the current study pulmonary moniliasis was diagnosed in three patients, one of whom showed a cavitary lesion roentgenologically. The fallacy of assuming monilial pathogenicity in instances of mere symbiosis has been emphasized by Kligman[31] and in the cases mentioned the evidence must be considered presumptive and not

conclusive. However, the pathogenic potentialities of *C. albicans* in exceptional circumstances are well-recognized. In general, mycologic studies and appropriate skin tests are indicated in cases with cavitary lesions when the more common etiologic agents are not discovered.

Parasitic Disease: Of the parasitic diseases involving the lung, those most likely to produce cavitation are amebiasis and hydatidosis. Sullivan and Bailey[32] have reported four cases of pulmonary infection with *Endameba histolytica*, three of which showed x-ray evidence of cavitation. Echinococcosis of the lung is usually evident roentgenologically as a solid lesion but may present the appearance of a cavity with or without a fluid level if the contents are evacuated surgically[33] or spontaneously.[4] Roentgenologic differentiation from tuberculosis may be difficult in some cases of pulmonary paragonimiasis. Although a "cystoidal" form of rarefaction may be encountered, instances of frank cavitation are decidedly uncommon.[34] Recovery of the ova from the sputum usually presents no difficulty in this disease. Cavitary lesions of the lungs due to parasitic forms other than those mentioned are infrequent.

Foreign Body: Aspirated foreign bodies, if not promptly extricated, may produce pathologic changes in pulmonary parenchyma and eventually result in cavitation. The most important factors in such instances would appear to be the nature of the foreign body, the duration of its presence, and the occurrence of infection. In the present series eight cases of intrapulmonary foreign body were encountered, of which four presented roentgen evidence of cavitation. The four foreign bodies associated with cavitation were a watermelon seed, timothy grass, a chicken bone, and a rubber eraser, while no cavitation occurred following inhalation of a peanut, a tooth with amalgam filling, timothy grass, and grass of undetermined nature. In general, the cavitary lesions were not pronounced. Since some of these patients either did not realize or had forgotten the fact of foreign body inhalation, often it was only upon pathologic examination of a surgical or autopsy specimen that the true etiology became apparent. Therefore, although the x-ray manifestations of cavitation are seldom striking, it is well to consider the possibility of inhaled foreign body when other etiologic factors are not discovered to explain the presence of cavitary disease.

Neoplasm: As with the previously mentioned entities, neoplasm involving the lung may similarly be associated with cavitation. Central necrosis, occurring in a rapidly growing tumor which has outstripped or mechanically compromised its available blood supply, may be reflected, roentgenologically and pathologically as cavitation. In addition, impairment of bronchial drainage by new growth may lead to infection, abscess-formation, and excavation. In view of this, cavitation might be anticipated less frequently with benign than with malignant pulmonary tumors and when seen with the former would more likely be the result of distal suppuration than of excavation of the tumor itself. Such has proved to be the universal experience and bronchial adenoma, the most "benign" pulmonary neoplasm,

is not generally considered an excavating lesion, although even here cavitation has been reported.[19] Malignant neoplasms, in contrast to benign, are much more frequently associated with cavity-formation (Figures 4 and 5). Of the 26 patients with bronchogenic carcinoma included in the present series, 16 showed excavation. According to histologic type, cavitation occurred in nine of 15 cases of undifferentiated carcinoma, six of nine cases of squamous cell carcinoma, and one of two cases of adenocarcinoma. Its occurrence in terminal bronchiolar ("alveolar cell") carcinoma[35] and adenoacanthoma[36] has been described. Gray and co-workers[37] indicate that the frequency of cavity-formation increases with the histologic grade of malignancy and that excavation is more prone to develop in the large, widely infiltrating, peripheral type of tumor. The prognostic and therapeutic importance of distinguishing malignant neoplastic cavitation from that due to other causes has been stressed by many authors.[38, 39, 40, 41, 42] Prescinding from the controversy as to whether Hodgkin's disease of the lung can exist as a primary lesion or is secondary to mediastinal involvement, occasionally cases are seen clinically in which the process apparently originates within pulmonary parenchyma. In such instances x-ray films may show evidence of cavitation.[19, 43, 44] Pathologically, Hodgkin's disease

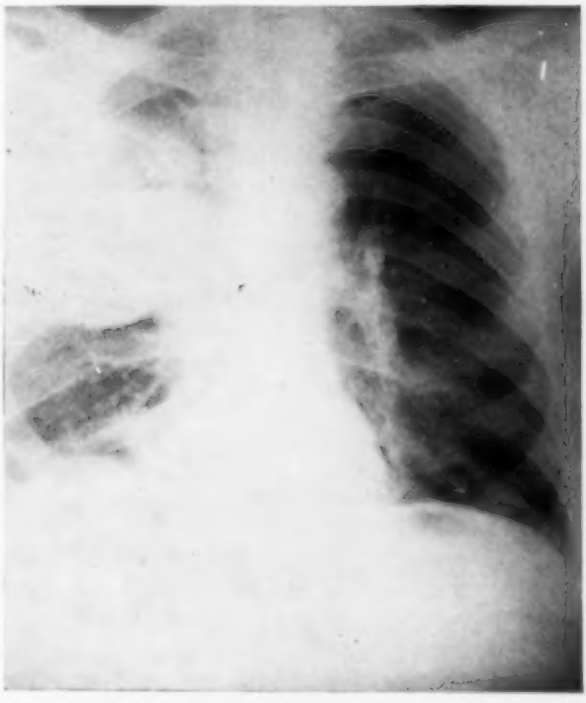

FIGURE 4: Bronchogenic Carcinoma. Huge fluid-containing cavity of the right lung in a 64 year old man with well-differentiated squamous cell carcinoma. (Courtesy of Department of Thoracic Surgery, U. S. Naval Hospital, St. Albans, N. Y.).

occurring in other organs is not characterized by necrosis and cavity-formation but when involving lung tissue may progress to excavation, presumably because of the peculiar anatomic characteristics of this organ.[44] According to Ritvo,[45] cavitation may be an uncommon manifestation of any of the lymphomas when involving pulmonary parenchyma. In a more recent report, Cooley *et al.*[46] have described cavitation in three of nine cases of primary lymphoma of the lung. As with primary malignancy, cavitation may be seen in secondary lesions of the lung as well,[47] but such cavities occur less frequently and generally are smaller in size (Fig. 6). In the present clinical material there were nine cases of secondary adenocarcinoma (eight primary in the female breast, one in the pancreas). Of these, five showed x-ray evidence of excavation. Such cavities, however, were far less striking than those seen in the patients with bronchogenic carcinoma. Cavitation developed in one patient with mediastinal teratocarcinoma which had invaded the lung. Three were encountered with pulmonary metastasis from non-epithelial malignancies (malignant giant cell tumor, lymphosarcoma, and synovioma) and none showed cavitation. Cavity-formation in secondary sarcomatous lesions of the lung is uncommon but has been observed.*[48, 49] Pulmonary cavitation may rarely complicate myelogenous leukemia.[50]

Infarction: Probably because of the dual blood supply of the lung, necrosis with cavitation is not often observed in uncomplicated pulmonary infarction but may occur if such lesions suppurate.[4, 51] Even in the absence of gross infection, however, cavitation may occur.[52] Two instances of pulmonary infarction are included in the present series, in one of which x-ray evidence of rarefaction in the infarcted area was noted, presumably due to infection with non-hemolytic *Streptococcus*. Excavating infarcts from septic emboli occur but such lesions might more properly be considered metastatic abscesses than suppurating infarcts.

Allergy and Collagen Disease: Attention has recently been directed to the association of cavitation with pulmonary pathology of allergic origin, Elkeles and Butler [53] describing a case of Loeffler's syndrome in which x-ray films demonstrated transient cavitation at the site of an apical pulmonary infiltrate. Less benign are the excavating lung lesions which may be observed in polyarteritis.[54, 55] Cavity-formation in such instances is believed to result from extensive infarction due to the arterial lesions. Acute arteritis of pulmonary vessels with resultant ischemic necrosis has similarly been invoked by Tumulty [56] to explain the occasional occurence of pulmonary cavitation in systemic lupus erythematosus. Cavity-formation in Wegener's granulomatosis [57] may have a similar pathogenesis.

Pneumoconiosis: Of the several pneumoconioses silicosis is the only one pertinent to the present investigation. Because of the high incidence of

*The case reported by Dolgoff and Hansen[48] is of especial interest. The patient was a 31 year old man with proven Ewing's sarcoma of the left femur who developed metastatic pulmonary lesions. During hospitalization a relatively large fragment of tissue was expectorated and found on pathologic examination to be consistent with Ewing's sarcoma. Chest x-rays secured immediately after the incident showed a cavity at the site of a previously noted metastatic nodule.

pulmonary tuberculosis among patients with this disease the roentgen demonstration of cavity-formation in such individuals has most often been considered *prima facie* evidence of silicotuberculosis. Even when autopsy fails to indicate the co-existence of tuberculosis, there is frequently great reluctance to exclude this etiology in the presence of cavitary lesions.[58] More recent work suggests that cavitation occurring in silicotic lungs does not necessitate a tuberculous factor and Vorwald[59] has shown that the fibrous connective tissue associated with "pure" silicosis may undergo necrosis and excavation in the absence of infection with *M. tuberculosis*. Wall[60] found non-tuberculous pulmonary cavitation in 15 of 100 autopsied cases of anthracosilicosis. Although the possible fallacy of considering anthracosilicosis a separate disease is realized, some workers[61] believe that coal dust, although not pathogenic in itself, alters the tissue response to silicon dioxide and that anthracosilicosis thus differs from silicosis. For the sake of completeness, therefore, it is listed in Table II as a distinct entity.

Sarcoid: Characterized by a histologic lesion which may often be distinguishable from that of tuberculosis only by the absence of necrosis, sarcoidosis is a systemic affection with an especial affinity for lung tissue. Because of its characteristic lack of necrosing tendencies, cavity-formation should not be anticipated. Although, as with silicosis, tuberculosis infection may later be superimposed, the demonstration of cavitary lesions does not necessarily implicate tuberculosis and it has gradually become apparent

FIGURE 5

FIGURE 6

Figure 5: Bronchogenic carcinoma. Postero-anterior tomogram of a 47 year old man with a large cavity in the superior segment of the left lower lobe. Malignancy is suggested by scalloping of the inner wall of the cavity. Pathologic examination of the resected lung showed squamous cell carcinoma.—*Figure 6:* Secondary Carcinoma. Postero-anterior chest x-ray fim of a 48 year old woman taken two years after right radical mastectomy for carcinoma. Metastatic disease is evident throughout both lungs and an excavating lesion is seen in the right lower lung field. The cavitation is more striking than is usually encountered in secondary carcinoma.

that uncomplicated sarcoidosis may exhibit cavitation.[62, 63]

Miscellaneous: As a curious and extremely rare cause of pulmonary cavitation, aortic aneurysm should be mentioned.[64] Also uncommon, pulmonary syphilis may be associated with cavitation, central necrosis in a gumma simulating cavitary tuberculosis.[12] Atelectasis as a cause of cavitation [65] is questionable but should be included among the rarer possible agents. Cacity-formation due to intensive radiotherapy is discussed by Hardin,[43] who states that excavation noted with Hodgkin's disease of the lungs has occasionally been attributed to x-ray therapy and not to the initial disease.

In addition to the many foregoing entities which have been found to be associated with cavitary lesions of the lungs, there are several intrapulmonary conditions which may appear to demonstrate cavitation roentgenologically but which are not characterized pathologically by necrosis. In this group should be included pulmonary emphysema,[66, 67, 68] as well as congenital cysts and allied cystic lesions such as those seen in xanthomatosis.[69] Of intra-pulmonary origin but not of pathologic significance, vascular shadows may occur in such a fashion as to present the roentgen appearance of cavitation.[4] Although rarely described, trauma to lung parenchyma may produce lesions which resemble cavities on x-ray films. This has been accomplished experimentally by Moolten [70] and its clinical occurrence documented by Balchum and Zimmerman.[71]

As noted in Table I, a variety of extra-pulmonary entities may simulate cavitary lung disease roentgenologically. Among these are encapsulated empyema [72] and loculated pneumothorax. Diaphragmatic hernia has been reported to present the roentgen characteristics of pulmonary cavitation [73] and in the present series a similar case was encountered. Pseudo-cavitary pulmonary lesions have been described on x-ray in patients with uremia.[74] Costal peculiarities may also be responsible for pseudo-cavity and according to Golden[4] an unusual distribution of calcium in the costal cartilage of the first rib may resemble cavitation on x-ray film. Finally, film artifact may result in a spurious "cavity."

Differential Roentgen Diagnosis

Thus far data have been presented to indicate the wide variety of nontuberculous conditions in which pulmonary cavitation, real or apparent, may occur. Only slight reference has been made to the differential roentgen diagnosis of the various lesions. Size and location of cavity, characteristics of its wall, and numerous other factors are of importance in suggesting the etiology. However, since "the roentgenographic diagnosis in itself is not conclusive",[12] differential x-ray characteristics have not been stressed. Studying a series of cavitary lesions, Diamond [75] tabulated roentgen aspects and the proved diagnosis, and was unable to establish a workable correlation. It is therefore evident that in any given patient with x-ray film evidence of cavitation a reliable etiologic diagnosis cannot be established on the basis of the roentgenogram alone but, as emphasized by Waring,[76] requires a critical appraisal of data secured by other means

as well. This is illustrated by a case in the present series. The patient, a middle-aged man, had been admitted to the hospital because of malaise, chest pain, and productive cough. X-ray films demonstrated a large apical cavity. The tuberculous nature of the latter was apparently confirmed by the finding of tubercle bacilli in the sputum. However, because the numerical concentration of the bacilli was thought to be less than would be expected with a cavity of the size noted, additional diagnostic measures were undertaken. These indicated the cavity to be due to a pyogenic lung abscess which promptly responded to appropriate therapy. The tubercle bacilli had originated from a small tuberculous focus, anatomically distinct from the abscess but possibly activated by it. Thus, even when the roentgenologic diagnosis of tuberculous cavitation is apparently confirmed by routine laboratory studies, it may often be well to investigate the possibility of another etiology.

SUMMARY AND CONCLUSIONS

The x-ray film demonstration of pulmonary cavitation is not equivalent to a diagnosis of tuberculosis since a wide variety of other conditions may produce or simulate cavitary disease of the lungs. Based upon a survey of the literature, a compilation has been made of all such entities. In a few of the intra-pulmonary diseases, included for completeness, authenticity has not been established beyond question. One hundred and forty-five cases of non-tuberculous clinical material observed at the Hermann M. Biggs Memorial Hospital, Ithaca, New York, during a 13-year period have been studied. One hundred and forty-one were instances of acquired non-tuberculous pulmonary pathology and in 98 of these cavitation or rarefaction was noted roentgenologically at some time in the course of the disease, an incidence of approximately 70 per cent. Three cases of lung cyst was encountered and all showed pseudo-cavitation on x-ray film. In one additional patient, the presence of a diaphragmatic hernia simulated cavitary disease of the lung roentgenologically. In determining the etiology of a cavitary process in any specific instance, the roentgen characteristics alone are not sufficiently circumscribed to permit final diagnosis and careful interpretation of diagnostic data obtained from other sources is essential. Since therapy of an excavating lung lesion may vary greatly depending upon the etiology, accurate diagnosis is important.

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RESUMEN Y CONCLUSIONES

La radiografía demostrativa de excavación pulmonar no equivale a un diagnóstico de tuberculosis ya que una gran variedad de padecimientos puede producir o simular afecciones cavitarias de los pulmones. Basándose en una revisión de la literatura, se hizo una compilación de todas las afecciones a que nos referimos. En algunas enfermedades intrapulmonares que se mencionan por ser completos ne so ha comprobado fuera de duda su autenticidad.

Ciento cuarenta y cinco casos de material clínico no tuberculoso observados en el Hospital Hermann M. Biggs Memorial de Itaca New York, se han estudiado por un período de 13 años. Ciento cuarenta y uno fueron casos de patología no tuberculosa adquirida y 98 de estas excavaciones o rarefacciones se notaron alguna vez en el curso de la enfermedad o sea una incidencia de 70 por ciento. Se encontraron tres casos de quistes pulmonares y todos mostraron aspecto radiológico seudocavitario. En un caso más la presencia de hernia diafragmática simulaba excavación pulmonar. No son suficientes las características radiológicas para determinar la etiología de cualquier caso especial y es esencial la obtención de otros datos de otras fuentes para el diagnóstico. El diagnóstico es importante desde el momento en que el tratamiento de la lesión excavada varía grandemente según su naturaleza.

RESUME

La certitude radiologique d'une caverne pulmonaire n'implique par forcément le diagnostic de tuberculose, puisqu'une grande variété d'autres affections peut produire ou simuler une cavité du poumon. Les auteurs ont fait la revue générale de toutes ces entités, basée sur une étude de la littérature. 145 cas de maladies non tuberculeuses ont été étudiés à l'Hôpital Hermann M. Biggs Memorial, Ithaca, à New York, pendant une période de 13 ans. 141 cas furent des exemples d'affections pulmonaires non tuberculeuses acquises. Dans 98 d'entre elles, on nota des excavations ou des zones de clarté à un moment de l'évolution de la maladie, soit approximativement 70% de cavités radiologiques. 3 cas de kystes pulmonaires furent rencontrés et tous donnèrent sur le cliché une image de fausse cavité. Chez un malade, une hernie diaphragmatique simulait radiologiquement une affection cavitaire du poumon. En déterminant l'étiologie du processus cavitaire de chaque cas particulier, les caractères radiologiques à eux seuls ne sont pas suffisants pour affirmer le diagnostic. Une interprétation réfléchie des éléments de diagnostic, obtenus à partir d'autres recherches est indispensable. Comme la conduite thérapeutique, en présence d'une lésion pulmonaire excavée, peut varier grandement selon l'étiologie, il est important d'en établir le diagnostic précis.

ZUSAMMENFASSUNG UND SCHLUSSFOLGERUNGEN

Der durch eine Röntgenaufnahme geführte Nachweis einer pulmonalen Cavernenbildung ist nicht gleichbedeutend mit der Diagnose einer Tuberkulose, da eine grosse Vielzahl anderer Krankheitsbedingungen zu cavernenartigen Lungenkrankheiten führen oder sie vortäuschen kann.

Auf der Grundlage einer Literaturübersicht wurde eine Zusammenstellung aller solcher Vorkommnisse gegeben. Bei einigen wenigen intrapulmonalen Krankheiten, die der Vollständigkeit halber mit aufgeführt sind, steht die Glaubwürdigkeit nicht ganz ausser Frage. Es wurden 145 Fälle eines nichttuberkulösen klinischen Materials untersucht, die im Hermann M. Biggs Memorial Hospital, Ithaca, New York, im Verlauf von 13 Jahren zur Beobachtung kam. 141 Fälle hatten erworbene nichttuberkulöse pathologische Lungenveränderungen und bei 98 von ihnen

wurde eine Cavernenbildung oder Aufhellung zu irgend einem Zeitpunkt im Krankheitsablauf röntgenologisch bemerkt mit einer Häufigkeit von ungefähr 70%. 3 Fälle von Lungencysten wurden gezählt und alle zeigten eine Pseudo-Cavernisierung auf dem Röntgenbild. Bei einem weiteren Patienten täuschte das Vorliegen einer Zwerchfellhernie röntgenologisch eine cavernöse Lungenerkrankung vor. Für die Bestimmung der Aetiologie eines cavernösen Prozesses in jedem Einzelfall liegen die röntgenologischen Merkmale allein nicht genügend fest, um eine endgültige Diagnose zu erlauben, und eine sorgfältige Deutung der aus anderen Unterlagen gewonnenen diagnostischen Daten ist wesentlich. Da die Therapie einer höhlenbildenden Lungenveränderung weitgehend verschieden sein kann in Abhängigkeit von der Aetiologie, ist eine sorgfältige Diagnose von Wichtigkeit.

References will appear in reprints.

Glucose Level in Pleural Fluid As a Diagnostic Aid

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Introduction

Clinicians for years have searched for some diagnostic means by which the number of cases of "Idiopathic Pleural Effusions" could be definitely identified as to etiology. Various tests upon the fluid removed have been proposed. Some have used the serology or pleural protein levels; others, the cellular morphology or glucose levels of the pleural fluid. Nearly all investigators have come to the conclusion that the only definitive diagnostic finding is the presence of neoplastic cells or the causative organism in the fluid removed. A few others, however, have felt that a determination of the glucose level in the fluid is an aid in differentiating tuberculous effusions from other types. The percentage of pleural fluids positive for acid fast bacteria has varied from 35 to 80 per cent, depending upon the series considered. Thus from 20 to 65 per cent remain in the idiopathic classification. They are so classified because: 1) They have a positive tuberculin skin test with negative fungal tests (some may have both types positive). 2) Usually they are below the average tumor age group. 3) Guinea pig inoculations and cultures of the pleural fluid for acid fast bacteria or other bacteria are negative. 4) The fluid is an exudate and not a transudate.

The problem of "T. B." or not, is not a purely academic one, for there are more than adequate statistics available to show that 30-40 per cent of patients with pleural effusions, if untreated, will within the following five years develop evidence of pulmonary or extra-pulmonary tuberculosis. It has also been proven that this 30-40 per cent morbidity could be reduced to 7-13 per cent with only four months of bed rest treatment. (The majority of all studies were done in the pre-chemotherapy era). The clinician is thus faced not only with the question of diagnosis, but also with the matter of whether or not to treat the patient. Once he has decided to treat the so-called "Idiopathic Effusion" as tuberculous, he has committed him to a number of months of chemotherapy and bed rest. However, with the poor prognosis for such a high percentage of untreated cases, many physicians feel obliged to treat both the proved and unproved cases as tuberculous. For the patients who do not have tuberculosis, but are treated as such, this represents a tremendous loss both to them and their immediate families. If it is the wage earner involved, his future earning power can be markedly affected by his hospitalization and treatment. His ability to get adequate insurance coverage at a rate he can afford is impaired for a number of years after his treatment has been completed.

There is a definite need for some means by which the number of cases

*Bret Harte Sanatorium.

diagnosed as "Idiopathic Effusions" can be reduced and the etiology ascertained. By more careful bacteriological studies this might be accomplished. Centrifuging larger volumes of pleural fluid before culturing the sediment, multiple cultures, and guinea pig inoculation whenever possible, would undoubtedly give us more bacteriologically proved cases. But these methods are expensive and what is perhaps more important to the patient, time-consuming. If the pleural fluid glucose level were of diagnostic significance it would be of great value. The study of the glucose level in one hundred cases of pleural effusion in which etiology was proved is the purpose of this paper.

Review of Literature

Pinner and Moerke (1930)[1] in a comprehensive study of effusions occuring with and without pneumothorax treatment, concluded that neither chemical nor serological data afford, per se, dependable diagnostic or prognostic criteria. They also felt that there was no parallelism between blood levels of sugar and effusion levels. However, the highest effusion level that they obtained (124 mgm. per cent) was in a diabetic with an elevated fasting blood glucose level of 300 mgm. per cent. Going over the entire group of cases, one finds that this was actually the only effusion level above 85 mgm. per cent. All in this group had far advanced or moderately advanced pulmonary tuberculosis.

Gruner[2] reported that "pleural fluids in tuberculous patients always gave a negative Molisch reaction."

Nassau[3] in 1941 attempted to correlate the levels of pleural fluid glucose with the results of culture examinations for acid fast bacteria, and to use the levels prognostically. From his study, Nassau drew the following conclusions: 1) A high sugar content in a pleural effusion (generally averaging about 70 mgm. per cent) was a favorable sign, indicating in all probability that the effusion would be a sterile one (negative culturally for acid fast bacteria). However, if the effusion were found to contain M. tuberculosis organisms, absorption was to be expected within a reasonable length of time (six to eight weeks). 2) A low sugar content (under 50 per cent) almost invariably indicated the presence of tubercle bacilli in the effusion, and that absorption would be delayed or that an empyema would ensue.

Gelenger and Wiggers[4] became interested in the problem of pleural fluid glucose levels in tuberculosis because of the characteristic low sugar levels found in the spinal fluid in tuberculous meningitis. These investigators, with 33 cases of effusions, went a step farther than Nassau. They compared the blood level of glucose with the pleural level at the time of the thoracentesis. Gelenger and his co-worker were able to prove the etiology in 31 out of 33 of the cases. They divided the cases into three groups depending upon the pleural sugar level found. Group I, consisting of eight cases, had a glucose level in the fluid of below 30 mgm. per cent. Group II had a level between 30 and 60 mgm. per cent; while Group III cases were all above 60 mgm. per cent. Group I had pleural sugar levels of up to 25.1 mgm. per cent and concomitantly studied venous blood glucose

levels of 82 to 142 mgm. per cent. In all of these cases tubercle bacilli were found. Group II had four patients, in which the level varied from 30.3 to 39.5 mgm. per cent, with an average level of 36.3 mgm. per cent. Blood glucose levels were from 104 to 122 mgm. per cent. In two of the four cases, tubercle bacilli were found at a later date. Of the remaining two, one was found to have a bronchogenic carcinoma, and no diagnosis was made in the other. The largest number of cases fell into Group III, with 21. Here the pleural fluid glucose levels were between 62 and 140 mgm. per cent, with blood levels of 70 to 166 mgm. per cent of glucose. No active tuberculous disease was found to be present in this group. The conclusions that these authors drew, were as follows: 1) Pleural sugar levels may be useful in obscure sero-fibrinous pleural effusions. 2) The lower the pleural sugar level, the greater the possibility of it being on a tuberculous basis. 3) All the tuberculous cases were found to be at a level below 39.5 mgm. per cent. Therefore, a finding of 30 mgm. per cent or less in the pleural fluid should be diagnostic of tuberculosis, and the organisms assiduously looked for. 4) Borderline cases of 30 to 60 mgm. per cent should be considered tuberculous until proved otherwise. 5) If the level is above 60 mgm. per cent it is unlikely that the cause of the effusion will be tuberculosis.

In 1951 Martinez[5] published an article covering 25 cases. He states that all of the 15 effusions of bacteriologically proved tuberculous etiology showed a pleural fluid glucose level of below 40 mgm. per cent. Non-tuberculous conditions on the other hand, such as cancer or pneumonia, gave glucose levels that paralleled the blood levels.

It is of interest to note that Martinez' results paralleled Gelenger's to some degree. He considered the significant level to be 40 mgm. per cent while Gelenger found all of his positive (tuberculous) cases to be below 39.5 mgm. per cent. Whereas both Gelenger and Martinez found somewhat similar results, Nassau's were entirely different, for he found positive cultures up to 84 mgm. per cent, with a range of from 15 mgm. to 84 mgm. per cent, and an average of 49.25 mgm. per cent.

Drazilova,[6] in 1954, reported a series of cases in which he determined the level in pleural effusions. He suggested that low values of 8 to 94 mgm. per cent were to be considered as typical of tuberculosis. High values of 100 mgm. per cent or over were never found in tuberculous effusions, but were noted quite often in effusions due to neoplasms. However, there was no comparison made of the effusion level and the concurrently run blood glucose levels.

Results of Present Study

The group that we are presenting consists of 100 patients with pleural effusions. The majority of these cases was admitted to a large county general hospital (720 beds), and to a tuberculosis sanatorium (270 beds), plus some that have been admitted to a Military Installation Chest Center. We have taken only patients in whom a definite bacteriological or pathological diagnosis was made. All of the patients considered had positive Mantoux skin tests (PPD was used for testing).

TABLE I
DIAGNOSES IN NON-TUBERCULOUS EFFUSIONS

	No. of Cases
Primary Carcinoma of the Lung	8
Cervical Carcinoma with pulmonary metastases	1
Bile Duct Carcinoma with pulmonary metastases	1
Bacterial Pneumonia (with organisms found)	2
Coccidioidomycosis	2
Histoplasmosis	1
"Mesothelioma"	1

Age, Sex, and Race: Ages of the patients studied ranged from 11 to 65 years. The ratio of males to females was 2:1, whereas the ratio of males to females with active pulmonary tuberculosis at the sanatorium during this same period was 4:1. The differences in the percentage of the different racial groups in our study as compared with the others, results from the prevalence of certain groups in the general population of this area. Fifty patients were Caucasian, 32 Oriental, ten Negroes, and eight Mexican. Our classification is admittedly an arbitrary one. Negroes made up only 1/10 of the total group. This, of course, is contrary to the high percentage of Negroes seen in Military Chest Centers.

Location of Fluid: Fluid was on the right side in 55 cases; the left side in 40, and was bilateral in five. Different authors have reported anywhere from 3 to 10 per cent bilateral effusions. However, there is a good deal of confusion in that some are referring to "Idiopathic Effusions," while others are referring to only those positive for acid fast bacteria. The highest percentage of bilateral effusions in the series reviewed occurred in the so-called "Idiopathic Group."

Blood Glucose Levels: All of the patients studied had normal fasting venous blood glucose levels (80 to 120 mgm. per cent). Another group who had abnormally high levels will be presented in another paper at a later date. However, we can point out at this time that a marked elevation in the blood sugar will definitely increase the pleural fluid level. For this reason it is necessary that fasting blood levels be ascertained concurrently with the pleural levels; to depend on this test in an uncontrolled or even controlled diabetic might result in a mistaken diagnosis.

Pleural Fluid Levels: As shown in the graph, there were 70 cases in which acid fast bacteria were found by some means in the effusion. These had an average pleural fluid glucose level of 26 mgm. per cent, with a low of 10 mgm. per cent and a high of 48 mgm. per cent. We found that the tuberculous empyemas had the lowest average levels, with an average of 15 mgm. per cent, and a range of 12 to 22 mgm. per cent. Effusions which were straw colored exudates and which were positive for acid fast bacteria on concentrated smear were close to the empyema levels, with an average of 17 mgm. per cent and a range of 14 to 20 mgm. per cent.

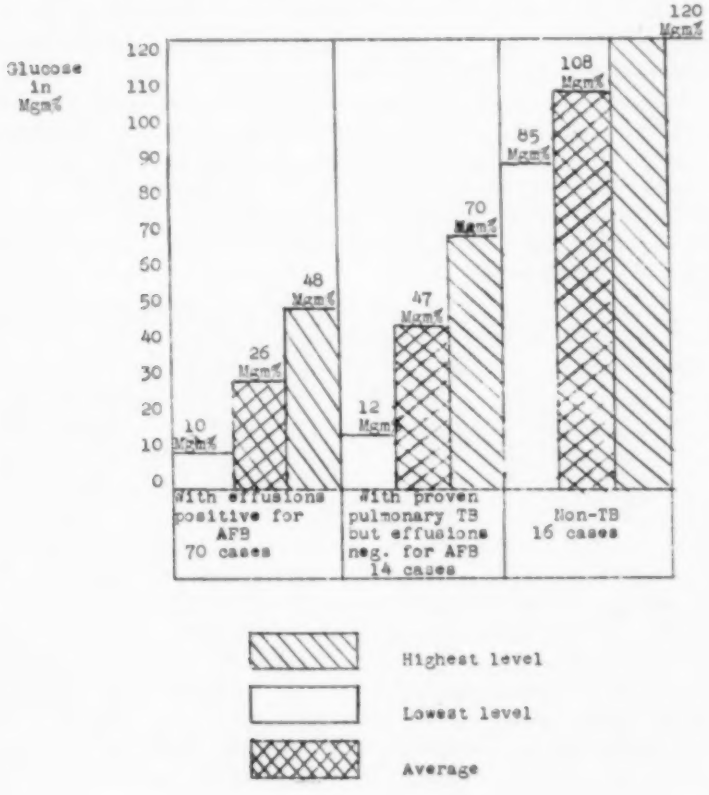
There were 14 patients (graph) in whom the pleural effusions were sterile, but who had proven parenchymal tuberculosis. This was proved by obtaining positive cultures of sputum or gastric contents or by surgical exploration. This group had a high of 70 mgm. per cent and a low of 12 mgm. per cent, with an average of 47 mgm. per cent.

In the remaining 16 cases, the pleural glucose levels were all above 70 mgm. per cent, the highest level seen in proved tuberculosis. The lowest level actually found in this non-tuberculous group (graph) of 16 cases was in a patient with lobar pneumonia. She was found to have a level of 85 mgm. per cent. The other 15 non-tuberculous cases had a range of from 90 to 120 mgm. per cent. The diagnoses are as listed in Table I, and were proved by surgery or autopsy.

Case Reports

Two cases, both of whom had non-tuberculous effusions but who were admitted because of the possibility of tuberculosis, are of particular interest in this series.

Case 1: A 65 year old white woman gave a history of working on a tuberculous ward at a General Hospital. Her lesion was picked up on a routine x-ray film survey and



was located in the right upper lobe. She gave a past history of having a cervical carcinoma removed some five years before, and at this time there was no evidence of recurrence. Because a sputum culture was positive for acid fast bacteria, she was admitted to the sanatorium and placed on standard antituberculous chemotherapy. After six months of treatment, and with no further positive sputums found, she developed pleural effusion on the right side. Glucose level in this fluid was 120 mgm. per cent and blood glucose was normal. 500 cc. of fluid was centrifuged and Papanicolaou stain done. Malignant cells were found in the specimen. A few days later she developed an effusion on the left side. An autopsy done at a later date revealed metastatic adenocarcinoma with no evidence of active tuberculosis found in either lung field.

Case 2: A 43 year old white woman was transferred to the sanatorium with a diagnosis of tuberculous pleurisy with effusion or idiopathic pleural effusion. She was not ill on admission but had had a slight fever on her initial entrance into the general hospital. She reacted three plus to second strength PPD, with negative coccidioidin and histoplasmin skin tests. Gastric lavages and several tracheal washes were negative for acid fast bacteria. She was classified as having "Idiopathic Effusion" but treated for tuberculous effusion with chemotherapy and bed rest. The pleural fluid disappeared without further taps. Suddenly, although under adequate chemotherapy, fluid reappeared on the same side (left). There was no fever, chills, or loss in weight. Except for slight pressure symptoms in the left chest, she was essentially asymptomatic. At this time a pleural glucose level was reported to be 116 mgm. per cent. (No glucose level was obtained on the initial fluid which was removed before transfer to the sanatorium). A repeat thoracentesis a few days later gave a level of 120 mgm. per cent with normal venous level. Papanicolaou stain of centrifuged sediment from some 600 cc. of straw colored exudate was reported positive for malignant cells. Thoracotomy was done, and the pleura was found to be studded with small nodules and grape-like clusters. These were biopsied and a small peel decorticated. Pathological finding was consistent with malignant "mesothelioma."

Conclusions

Unlike Gelenger and Wiggers who obtained cultures positive for acid fast bacteria only in cases with fluid glucose levels up to 39.5 mgm. per cent, we were able to find positives up to 48 mgm. per cent. If one includes those with abacillary effusions but with proved parenchymal tuberculous lesions, we were able to find proof of tuberculosis at levels of up to 70 mgm. per cent. However, we were still considerably below Nassau's findings, in which he obtained positive cultures in cases with levels up to 97 mgm. per cent.

From our studies, we must concur with Gelenger and Wiggers that the lower the glucose level in the effusion, the better are the chances that it is on a tuberculous basis. This is especially true if the level is at about 26 mgm. per cent or lower. As noted above, all positive concentrates for acid fast bacteria were obtained from cases with pleural fluid levels of between 14 and 20 mgm. per cent.

In all of the effusions we studied, as much fluid as possible was removed, and at least 500 to 600 cc., if it was available, was sent to the laboratory. We requested that routinely all of it be centrifuged and the sediment cultured or inoculated into guinea pigs. If possible, several cultures were made and guinea pig specimens inoculated concurrently either from one specimen or from several specimens removed at close intervals. We feel that by this method we increased the number of positive cultures by at least 30 to 40 mgm. per cent over that obtained with a single culture or inoculation of a small amount of fluid.

It is our feeling that a patient with a level of above 80 mgm. per cent (with normal venous blood glucose level) should not be automatically

placed in the "tuberculous until proved otherwise" group and treated with chemotherapy, at least not until every other means of obtaining a positive diagnosis has been exhausted. Other means should include either pleural biopsy or thoractomy, and it is our impression that a thoractomy incision which is adequate for visualization is better than a simple pleural biopsy. We have not seen a sufficient number of pleural biopsies to make us feel certain of its adequacy unless one accepts a negative pleural biopsy as an indication that no active tuberculosis is present. Certainly we have found nothing to indicate that the pleural glucose level should be used as the sole criterion. Other laboratory aids of proved diagnostic value should be relied upon primarily, with the glucose level adding supplemental or confirmative information. We have several reasons for not being completely convinced of the accuracy of the test. Among these reasons are: 1) Pleural fluid specimens with known glucose levels may be reported as having varying amounts of glucose when the tests are done by different technicians. Therefore, how can the test be used as a fine differentiating point when 3-5 mgm. per cent would place one case in the tuberculous category and the other in the non-tuberculous? 2) Unless the glucose level is done within a short time after thoracentesis, the determination is inaccurate. 3) It is our impression (although the study is not complete) that varying venous sugar levels will bring about varying pleural levels. 4) Our series of non-tuberculous effusions is small, consisting of only 10 cases.

SUMMARY

1. Results of pleural fluid glucose determinations in 100 cases with normal blood sugar levels are described.

2. The conclusion is made that pleural fluid glucose of 26 mgm. per cent or lower are highly suggestive of tuberculosis as the cause of the effusion. In cases with levels of 80 mgm. per cent or higher, one should be suspicious of a non-tuberculous etiology and perhaps should consider early thoractomy in order to establish the diagnosis.

3. Although pleural fluid glucose levels are a valuable tool, they should not be relied upon as the sole diagnostic criterion.

RESUMEN

1. Se describen los resultados de la determinación de la glucosa en el líquido pleural en 100 casos con glucemia normal.

2. Se concluye que la presencia de glucos en el líquido pleural a razón de 26 mgrs. o menos por ciento, es altamente sugestiva de que la tuberculosis sea la causa del derrame.

En casos con 80 mgrs. o más debe uno sospechar que no se trate de tuberculosis y probablemente considerer el hacer una toracotomía inmediata para establecer el diagnóstico.

3. Aunque los niveles de la glucosa en los líquidos pleurales sean un valioso recurso, no se debe confiar en ellos solamente para el criterio diagnóstico.

RESUME

1. Les auteurs décrivent les résultats des déterminations du glucose du liquide pleural dans cent cas chez lesquels le taux de sucre sanguin était normal.

2. Ils arrivent à la conclusion que les taux de glucose du liquide pleural de 26 mmgr. pour cent ou moins sont hautement évocateurs d'une origine tuberculeuse de l'épanchement. Dans les cas où les taux atteignent 80 mmgr. pour cent ou plus, on doit suspecter une étiologie non tuberculeuse et peut-être envisager une thoracotomie précoce pour établir le diagnostic.

3. Bien que le taux de glucose du liquide pleural soit donc un renseignement de valeur, il ne doit pas être considéré comme le seul critère diagnostique.

ZUSAMMENFASSUNG

1. Beschreibung der Ergebnisse der Glukose-Bestimmung der Pleuraflüssigkeit an hundert Fällen mit normalen Blutzuckerspiegel.

2. Es wird der Schluss gezogen, dass Glukose-Spiegel der Pleuraflüssigkeit von 26 mg. % oder weniger hochgradig verdächtig sind auf eine Tuberkulose als der Ursache des Ergusses. Bei Fällen mit einem Spiegel von 80 mg. % oder mehr muss man eine nicht-tuberkulöse Aetiologie vermuten und vielleicht eine frühzeitige Thorakotomie in Erwägung ziehen, um die Diagnose zu stellen.

3. Auch wenn der Glukose-Spiegel der Pleuraflüssigkeit ein wertvolles Hilfsmittel darstellt, darf man sich doch nicht darauf als einzigem diagnostischen Prüfstein verlassen.

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Histoplasmosis on a Small Tuberculosis Service in a General Hospital

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The recognition of mycotic infections of the lungs is particularly desirable on a Tuberculosis Service so that patients with mycotic infections alone may avoid unnecessary exposure to tuberculosis. When pulmonary mycosis and pulmonary tuberculosis are present at the same time and are recognized, appropriate therapy for the mycotic infection may be added to the anti-tuberculosis treatment.

Pulmonary histoplasmosis, like pulmonary tuberculosis, may occur either as a primary type with little or no symptomatology, or as a reinfection type[1] which may result in progressive disease with fibrosis and cavity formation. As in tuberculosis, the primary infection results in hypersensitivity which may be demonstrated by the histoplasmin test. The hypersensitivity may remain for many years or possibly for life. The complement fixation test becomes positive, but is transitory and reverts in a period of months. There is an excellent discussion of the diagnostic significance of these findings by Smith.[2] A chronic progressive form of pulmonary histoplasmosis with cavity formation has been described by Furcolow and Brasher.[3] In this form of the disease positive sputum cultures for the fungus usually are obtained without difficulty. This type of pulmonary histoplasmosis resembles the fibrocaseous reinfection type of pulmonary tuberculosis. There is no present satisfactory specific treatment for histoplasmosis and the treatment of the chronic progressive form of the disease is similar in many ways to that of tuberculosis before the discovery of anti-tuberculosis drugs. Furcolow and Brasher found 7.2 per cent of 600 patients at a tuberculosis sanatorium had positive serology (complement fixation or precipitin) for histoplasmosis. Thirty-three per cent of these cases with positive serology had either a positive culture for *H. capsulatum* or organisms typical of *H. capsulatum* in tissues removed surgically or at autopsy.

The coexistence of pulmonary mycosis and pulmonary tuberculosis has been reported many times. Meleney[4] presented a series of cases in which pulmonary histoplasmosis and tuberculosis were present and active simultaneously. Three cases of pulmonary tuberculosis were reported by Larkin and Phillips[5] with a different coexisting pulmonary mycosis in each case. *Cryptococcus hominis* was present in one, *Coccidioides immitis* in the second and histoplasmosis in the third. All three ended

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fatally. Roger, Terry and Binford[6] reported two cases in which tuberculosis and histoplasmosis were associated with Hodgkin's disease. Six cases of coexisting active infection with *Coccidioides immitis* and *M. tuberculosis* were described by Stein.[7] A case of Addison's disease associated with disseminated histoplasmosis and pulmonary tuberculosis was recently reported by Fitzpatrick and Reuber.[8] A case which was admitted to the tuberculosis service at the Veteran's Administration Hospital, Wadsworth, Kansas in September 1954 was diagnosed as pulmonary tuberculosis when a chest x-ray film showed bilateral fibrocavernous disease and sputum cultures were reported positive for *M. tuberculosis*. Three and one-half months later a postmortem examination showed coexisting disseminated histoplasmosis with involvement of the adrenals, kidneys, trachea, lungs, spleen, liver, bone marrow, and pancreas. Two other patients admitted to the tuberculosis service during 1955 were found to have pulmonary mycosis without tuberculosis. Only of these had cavitary histoplasmosis of the lungs and the other had sporotrichosis. This case will be the subject of a separate report at a later date.

Case 1: W. Y., a 66 year old white male gas welder, was admitted to the tuberculosis service at Wadsworth, Kansas on September 21, 1954. Pulmonary tuberculosis had been diagnosed early in September with acid fast bacilli in sputum smears and extensive disease revealed by x-ray film. This was confirmed by repeatedly positive sputum cultures for tuberculosis. There was a history of progressive weakness, anorexia and loss of about 30 pounds during the eight months preceding hospitalization. There had been slight hoarseness for six weeks preceding admission. He had syphilis and was treated in 1927 and in 1954.

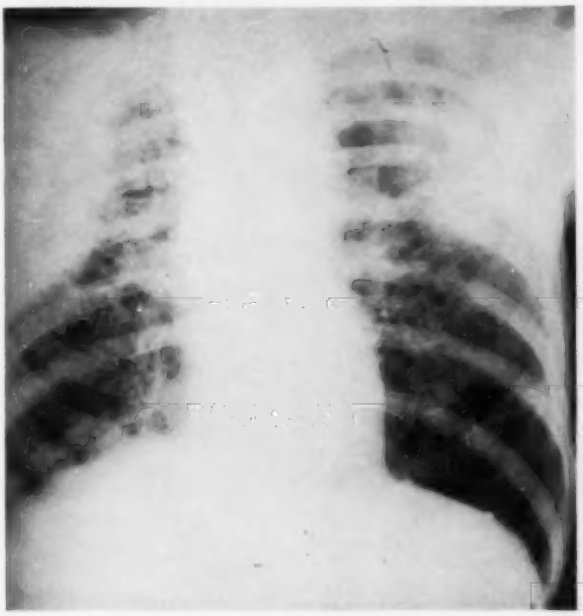

FIGURE 1A: Admission film (November 24, 1954) showing extensive bilateral, fibrocavernous disease.

FIG. 1B

FIG. 1C

FIG. 1D

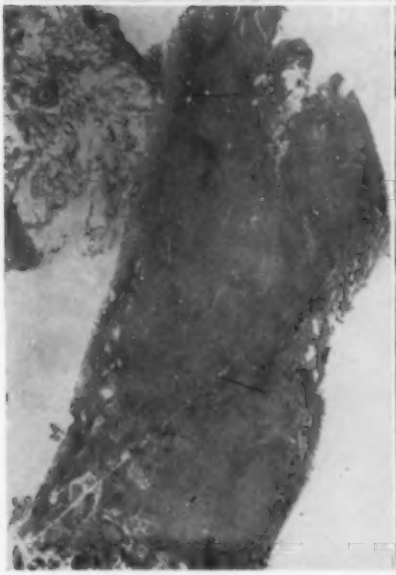

FIG. 1E

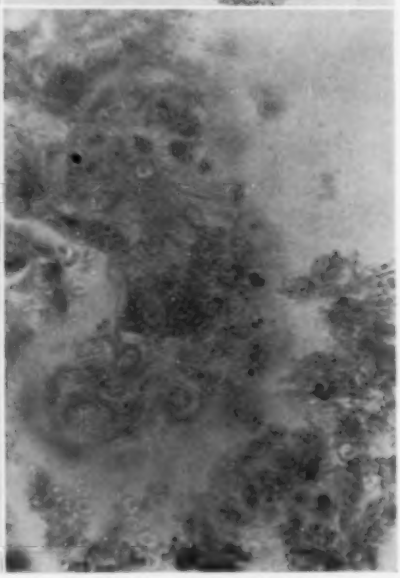

Figure 1B: Lung section through a tuberculous cavity. *Figure 1C:* Lung section showing acid fast bacilli but no histoplasma. *Figure 1D:* Lung section resembling solid organ due to reticulum cells filled with histoplasma. *Figure 1E:* Higher magnification showing masses of histoplasma but no tubercle bacilli.

He was emaciated and appeared acutely ill. He weighed 100 pounds compared with a normal weight of 130 pounds. His temperature was 97.2° F.; pulse 80; respiration 22 and blood pressure 100/74. The chest expansion was bilaterally limited. There was diminished vocal fremitus over both lungs and moist rales over all portions of both lungs. The liver was somewhat enlarged and there was mild edema of the ankles. The electrocardiogram and cardiac examinations were consistent with cor pulmonale and mild pulmonary hypertension.

The x-ray film of November 24, 1954 showed far advanced bilateral pulmonary disease involving all portions of both lungs with cavities on both sides. The base of the left lung was emphysematous (Figure 1A). A frontal planigram on December 7, 1954 confirmed the presence of cavitation in the upper portions of both lungs.

Six sputum smears and three cultures were reported positive for M. tuberculosis. The organisms obtained on September 23, 1954 were sensitiive to streptomycin and isoniazid but resistant to para-aminosalicylic acid. Blood serology (VDRL and Kolmer) were positive on admission. Skin tests using intermediate strength PPD and histoplasmin 1:500 were negative. Blood cultures were sterile. The blood hemoglobin decreased from 9.7 grams in September 1954 to 7 grams in December 1954. The red blood count fluctuated around 3,500,000. The white blood cell count on admission was 11,400, gradually decreasing and levelling off at 4,000 during the latter part of November 1954. The erythrocyte sedimentation rate gradually fell from 28 mm. per hour in September 1954 to 20 mm. per hour in December 1954. At various times the urine contained from a trace to 1 plus albumin. In December 1954 the urine showed a clumping of white and red blood cells and granular casts. The blood urea nitrogen gradually increased from 27 mg. per cent in October 1954 to 42 mg. per cent in December 1954.

Streptomycin one gram twice weekly, isoniazid 300 mgm. daily and para-aminosalicylic acid 12 grams daily were started on October 1, 1954 and continued with the exception of short interruptions until death. Penicillin 9,600,000 units was given because of the positive blood serology. After the first week of hospitalization he remained afebrile except for occasional temperature elevations to 99. Although there was slight improvement by chest x-ray film, he remained clinically unimproved and died three and one-half months after admission.

Necropsy: The body was that of a moderately well developed, poorly nourished, white man. A few small petechiae were present over both forearms. The mesenteric nodes were small and somewhat shotty. Enlarged para-aortic nodes measuring as much as 3 cm. in the long axis were located along side each renal artery. Extensive pleural adhesions were present bilaterally, particularly over the apices and upper lobes.

The right lung weighed 580 and the left, 680 grams. The most extensive pathology was found in the right upper and middle lobes which were markedly distorted as a result of extensive fibrosis. Small caseous lesions and cavitation were demonstrated. Cavities were present in the right upper and lower lobes, and left upper lobe. There were multiple superficial ulcerated zones in the lower portion of the trachea.

Both adrenal glands were plump and enlarged. The combined weight was estimated at 40 to 50 grams. Cut section showed almost complete replacement by pale, yellowish-gray tissue in which a few necrotic markings were observed.

The right kidney weighed 150 and the left 170 grams. The cut surface showed an ulcerative lesion involving several papillae in each kidney with extension of yellowish-gray tissue into the adjacent parenchyma. There were a few pale gray zones located just beneath the capsule of the left kidney. The cortico-medullary junction was distinct for the most part, although the cortex was pale and the glomeruli were not well visualized. The pelves and ureters were not dilated.

The spleen weighed 240 and the liver, 1,650 grams. Neither organ was remarkable grossly.

Sections taken from the two largest cavities (Figure 1B) showed evidence of tuberculosis with a ragged lining of granulation tissue in which epithelioid cells were prominent along with a few scattered giant cells. Acid-fast bacilli were demonstrated, but the periodic acid Schiff stain failed to color Histoplasma capsulatum. Sections from the small caseous lesions (Figure 1C) showed characteristic, well delineated tubercles with little evidence of activity at the periphery. Appropriate stains again demonstrated acid-fast bacilli but failed to show histoplasma. A single lung section resembled a solid organ (Figure 1D) due to the presence of innumerable reticulum cells whose cytoplasm was filled with histoplasma. At the periphery, the infected macrophages were seen to invade the wall of a small bronchiole and also to lie free within the lumen. The fungus stained readily with the PAS stain, and acid-fast organisms could not be demonstrated here (Figure 1E). Sections from the ulcerated trachea were filled with masses of histoplasma, and no tubercle bacilli could be found.

There was massive necrosis of the renal papillae with heavy infestation by Histoplasma capsulatum which also involved the peripelvic fat and extended into the cortex. Sections stained by the PAS method were colored deep red to the naked eye. A few fungi were demonstratd in the glomeruli, and in some instances were present in the tubules.

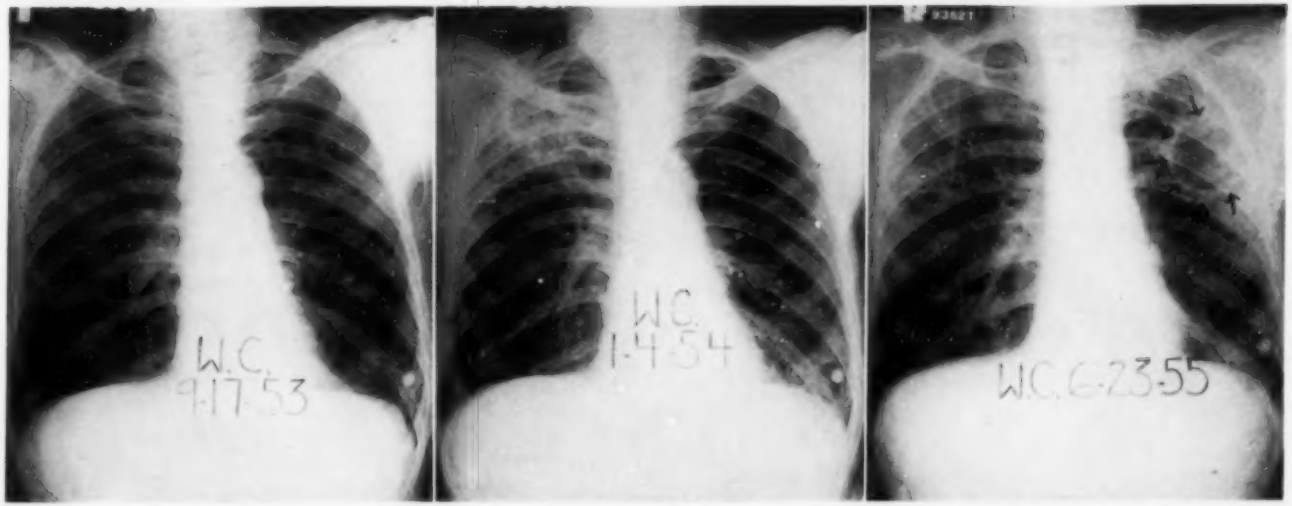

FIGURE 2A FIGURE 2B FIGURE 2C

Figure 2A: The earliest available film (September 17, 1953) showing disease in the upper right and in the lower left lung. *Figure 2B:* Dense infiltrate in upper right lung (January 4, 1954) resulting in the diagnosis of pulmonary tuberculosis. *Figure 2C:* Admission film (June 23, 1955) showing 6 cm. cavity in upper left lung.

Sections from each adrenal gland showed massive necrosis of the parenchyma. Histoplasma laden macrophages were so numerous that a slide stained by the PAS method appeared red to the naked eye. Small granulomatous lesions showing epithelioid-like and giant cells without necrosis were found in the spleen, liver, lymph nodes, pancreas, and a coronary artery. The bone marrow showed minimal involvement. The periodic acid Schiff stain demonstrated Histoplasma capsulatum in each of the above organs. Tubercle bacilli could not be found. Hiistoplasma capsulatum was cultured from an adrenal and from a kidney removed at the time of autopsy. The lung was not submitted for bacteriological examination.

Case 2: W. C., a 60 year old white, male, barber was admitted to this hospital on June 22, 1955 for treatment of lacerations resulting from a fall while under the influence of intoxicants. A routine chest x-ray film showed extensive bilateral pulmonary disease and a giant cavity in the upper left lung. When a history of previous treatment for pulmonary tuberculosis was elicited, he was transferred to the tuberculosis service. Pulmonary tuberculosis was first diagnosed on January 4, 1954. Four sputum and three gastric cultures obtained at that time were subsequently reported negative for acid fast bacilli.

There were several lacerations over the forehead. The temperature was 98.4° F., pulse 76 and blood pressure 112/68. He weighed 114 lbs. compared with his average weight of 140 lbs. The right pupil reacted sluggishly to direct light and there was a central lens opacity. Coarse rales were present over both hilar regions and over the upper left chest. No enlargement of the liver, spleen, or kidneys was detected.

The earliest available x-ray film of September 17, 1953 (Figure 2A) showed disease in the upper half of the right and in the base of the left lung. There was progression of the disease, shown on the x-ray film of October 26, 1953, throughout the remainder of the right and into the lower two-thirds of the left lung. There was a dense infiltrate in the upper right lung on January 4, 1954 (Figure 2B) when the diagnosis of pulmonary tuberculosis was made. No film was available between January 4, 1954 and June 23, 1955. During this interval the disease in the upper right lung became less dense and more scattered in appearance. Fibrocavernous disease appeared in the upper part of the left lung where an annular shadow measuring 6 cm. in diameter was visible (Figure 2C). A left lateral view taken on July 19, 1955 showed that the cavity was located somewhat anteriorly. Subsequently the annular shadow previously seen had decreased to a slit with a maximum diameter of 1 cm. before he left the hospital against medical advice.

Eleven sputum cultures were reported negative for tuberculosis. Eleven other sputum cultures were reported positive for Histoplasma capsulatum. Skin tests showed no hypersensitivity to PPD 1, coccidioidin 1:500 or blastomycin 1:100. Skin tests using PPD 2 and histoplasmin 1:500 did show hypersensitivity to both. Complement fixation tests for histoplasmosis were reported negative on four occasions. The red blood count increased from 3,600,000 on June 23, 1955 to 4,400,000 on January 18, 1956. The hemoglobin gradually increased from 10.5 gms. on June 23, 1955 to 12.6 gms. on February 20, 1956 just before discharge. The hematocrit changed from 38 VPC on June 23, 1955 to 43 on February 20, 1956. The erythrocyte sedimentation rate also gradually increased from 29 mm. per hour on June 23, 1955 to 37 mm. per hour on February 20, 1956. The serum albumin on admission was 2.6 gm. per cent and at discharge 3.7 gm. per cent. The serum globulin varied within the range of normal (2.6 gm. per cent to 3.4 gm. per cent). Blood serology for syphilis (VDRL) was negative.

Mycostatin was administered intravenously. The total dose was 3,577,500 units during a 38 day period between December 27, 1953 and February 2, 1956. Chilliness, substernal burning sensations, nausea, and transitory febrile reactions were frequent during and immediately following these intravenous injections. The drug was discontinued when he declined further intravenous therapy. He gained from 114 to 120 pounds during the eight months of hospitalization. At the completion of mycostatin therapy the only noticeable difference in the chest x-ray films was that the large annular cavity 6 cm. in diameter had changed to a narrow slit varying in width from 0.5 to 1 cm.

Discussion

We have found serological screening tests for histoplasmosis helpful in selecting cases whose sputum should be cultured for fungi. Since November 1955 we have participated in a serological survey conducted by Michael L. Furcolow for the United States Health Service in several hospitals in this area. Complement fixation tests for histoplasmosis have been done on 111 patients on the tuberculosis service at Veterans Administration Hospital, Wadsworth, Kansas. One hundred and seven of these

were reported negative, three were positive and one was anti-complementary. The sputum from one of the patients with positive serology for histoplasmosis yielded cultures positive for sporotrichum. Sputum from the other two were negative for fungi but positive for M. tuberculosis. It is possible that these two patients may have active histoplasmosis together with tuberculosis, but are not expectorating sufficient quantities of fungi to yield positive cultures. Complement fixation tests for histoplasmosis have been done routinely on each new admission to the tuberculosis service since we began our participation in the survey.

The diagnosis of pulmonary mycotic infection rests upon the demonstration of the etiologic organism. There are no characteristic radiologic findings which differentiate pulmonary mycotic infections from pulmonary tuberculosis. In an individual where the diagnosis of tuberculosis has not been definitely established, the differential diagnosis should include neoplasm and lung abscess, as well as mycotic disease.

SUMMARY

1. Since mycotic pulmonary infections are now relatively more frequent, their differential diagnosis is especially important on tuberculosis services because of their clinical and radiologic similarity to pulmonary tuberculosis.

2. Two cases with histoplasmosis are reported. One had repeatedly positive sputum cultures for Mycobacterium tuberculosis, and histoplasmosis was diagnosed only at postmortem by microscopic tissue examination with PAS stain and culture of Histoplasma capsulatum from autopsy material. The other had a giant cavity on admission and progressive disease on serial chest x-rays films over a period of nearly two years. Sputum cultures were positive for Histoplasma capsulatum and negative for Mycobacterium tuberculosis. Intradermal sensitivity tests were positive with both tuberculin and histoplasmin. Complement fixation tests were negative for histoplasmosis.

RESUMEN

1. Ya que las infecciones micóticas pulmonares son ahora relativamente más frecuentes, su diagnóstico diferencial es especialmente importante en los servicios de tuberculosis debido a su similitud clínica y radiológica con la tuberculosis pulmonar.

2. Se reportant dos casos de histoplasmosis. Uno tuvo cultivos positivos para Mycobacterium tuberculosis repetidos, y la histoplasmosis fué diagnosticada únicamente post-mortem mediante el examen microscópico histológico con tinción de PAS y cultivo de Histoplasma capsulatum del material de autopsia. El otro tenía una caverna gigante a la admisión y enfermedad progresiva evidenciada por el estudio radiológico seriado durante un período de cerca de dos años. Los cultivos de esputo fueron positivos para Histoplasma capsulatum y negativos para Mycobacterium tuberculosis. Las pruebas intradérmicas de sensibilidad fueron positivas tanto con tuberculina como con histoplasmina. Las pruebas de fijación del complemento fueron negativas para la histoplasmosis.

RESUME

1. Depuis que les mycoses pulmonaires sont devenues plus fréquentes, leur diagnostic différéntiel est particulièrement important dans les services de tuberculose, à cause de leur ressemblance clinique et radiologique avec la tuberculose pulmonaire.

2. Les auteurs rapportent deux cas d'histoplasmose. L'un présenta des cultures d'expectoration positives pour le bacille de Koch d'une façon répétée, et l'histoplasmose fut diagnostiquée seulement à l'examen microscopique des tissus post-mortem avec culture d'histoplasma capsulatum. L'autre cas présentait une cavité géante lors de son admission, et on vit l'affection progresser sur des clichés thoraciques en série pendant une période de près de deux ans. Les cultures d'expectoration furent positives pour l'histoplasma capsulatum et négatives pour le bacille de Koch. Les tests intradermiques furent positifs à la fois à la tuberculine et à l'histoplasmine. Les tests complémentaires de fixation furent négatifs pour l'histoplasmose.

ZUSAMMENFASSUNG

1. Nachdem Pilzinfektionen der Lungen gegenwärtig häufiger werden, wird auch ihre Differential-Diagnose von besonderer Wichtigkeit für die Tuberkulose-Abteilungen wegen ihrer klinischen und röntgenologischen Ähnlichkeit mit der Lungentuberkulose.

2. Es wird über 2 Fälle mit Histoplasmose berichtet. Bei dem einen lagen wiederholt positive Sputumkulturen von mycobacterium tuberculosis vor, und die Diagnose der Histoplasmose wurde nur auf Grund der Sektion gestellt durch mikroskopische Gewebsuntersuchungen mit PAS-Färbung und Kulturen von histoplasma capsulatum aus dem Sektionsmaterial. Der andere hatte eine Riesencaverne bei der Einweisung und eine fortschreitende Krankheitsform anhand der Röntgenbildreihe während eines Zeitabschnittes von nahezu 2 Jahren. Kulturen des Auswurfes waren positiv auf histoplasma capsulatum und negativ auf mycobacterium tuberculosis. Intracutane Empfindlichkeitsproben waren sowohl positiv auf Tuberkulin wie auf Histoplasmin. Komplement-Bindungsreaktionen auf Histoplasmose waren negativ.

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Apico-osteoplasty: A Modified Thoracoplasty Technique*

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Introduction

Since the advent of antimicrobial therapy, resectional surgery for pulmonary tuberculosis has become more popular.[1] With increased experience it has become apparent that space reducing procedures are often necessary following surgical removal of tuberculous cavitary or nodose disease.[2] The ideal procedure following pulmonary resection should fulfill the following requirements: 1) provision for complete obliteration of the vacant pleural cavity; 2) no need for insertion of foreign bodies; 3) effected with ease and rapidity so as to lessen the operating time, tissue trauma and blood loss; 4) absence of paradoxical motion, and 5) a good cosmetic result.

Although various methods for handling the troublesome pleural space have been employed, none of these have fulfilled all the aforementioned requirements.[1-7] A new technique, apico-osteoplasty, was therefore developed in our hospital, by one of us, (M. G.), and applied clinically. The operation consists in fashioning an osseo-muscular plate or "hard portion" with the second and third ribs and associated intercostal muscles. This is followed by extrafascial apicolysis with plication at the selected level.

Method

The standard posterolateral thoracotomy approach, entering through the periosteal bed of the retained sixth rib is used. Following resection, the remaining lung is inflated by the anesthetist using 10 cm. of H_2O pressure. At this point, the indications for a space filling procedure are evaluated. If necessary, it is performed as follows:

A. Musculo-osseous Flap or Hard Portion

This entails the creation of a moveable plate of the second and third ribs and associated intercostal musculature. A Finochietto rib spreader is left in place following the lung resection and the second and third ribs are approached intrapleurally and anteriorly, in close proximity to the costochondral junctions. Linear incisions, 3 cm. in length, are made through the pleura, thoracic fascia and periosteum. Segments of the rib measuring 1-2 cm. in length are removed subperiosteally. The rib spreader is then removed and the scapula is rotated lateralward to

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expose the external aspects of the ribs. Following a division of the appropriate insertions of the serratus posterior superior, similar segments of the second and third ribs are removed extrapleurally at their necks. The extrinsic muscle attachments of this plate, consisting of the posterior scalenus, serratus anterior and pectoralis minor are then divided. To insure full unrestricted mobility to the flap, the intercostal muscles of the second and third intercostal space are divided posteriorly, leaving intact the corresponding nerves and vessels. (Figure 1).

B. Apicopexy or Soft Portion

Apicolysis is then performed in an extrafascial plane. This is started by stripping the periosteum from the inferior surface of the first rib, Care is exercised in freeing the pleural dome from Sibson's fascia and the continuous subclavian vessels. This dissection is continued to a selected level, which is usually the aortic arch or azygos vein. The redundant apical pleura is imbricated upon itself and fixed to the mediastinal pleura using continuous or interrupted chromic catgut sutures. (Figure 2).

The lung is then fully expanded and chest closed in layers with two chest tubes connected to under water drainage.

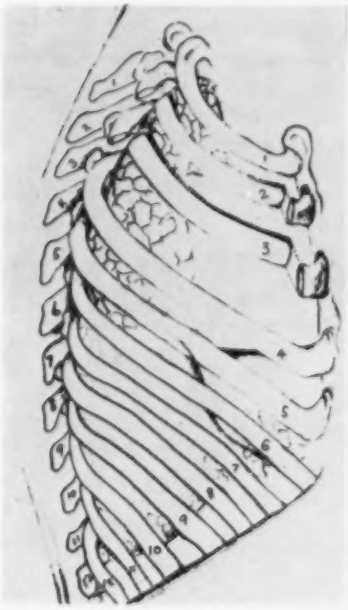

FIGURE 1

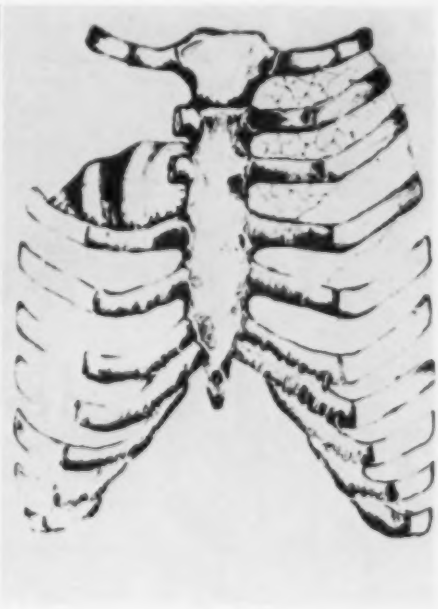

FIGURE 2

Figure 1: The second and third ribs are partially resected intrapleurally at the chondrocostal junction anteriorly; and extrapleurally close to the costal angle posteriorly.—*Figure 2:* Apicolysis is performed, the osseo-muscular plate is carried downward, the apical pleura imbricated and fixed to the mediastinal pleura with continuous or interrupted sutures.

TABLE 1

Name	Discharged	Admitted	Age	Onset	Classification of Disease	Operative Procedure	Complications	DEATH	SPUTUM[1]										PULMONARY FUNCTIONS			
									PRE-OP					POST-OP					PRE-OP		POST-OP	
									S	CUL	CAT	SM	INH	S	CUL	CAT	SM	INH	VC	MBC	VC	MBC
A. E.	3-10 1956	11-3 1955	26	Feb. 1955	Mod-Adv. Active	LLL+TP 28-Dec-55	0	0	0	1+	3+	1+	4+	0	0				4700	122	3660	66
J. T.	4-3 1956	9-16 1955	43	1943	Far-Adv. Active	Rul+SupSegRII + TP 10-Feb-56	Empyema and Fistula	0 0	50/ F	4+	+	3+	4+						2050	52		
B. M.	2-14 1956	10-23 1954	20	1948	Far-Adv. IN Active	Rul + TP 30-Sept-55	0	0	0	1+	0	0	0	0	0				1300	44		
B. Mc.	1-14 1956	5-7 1955	45	1952	Far-Adv. Active	Rul+SupSegRII + TP 19-Oct-55	0	0	2/ 50 F	4+	+	4+	4+	0	0				4420	97	3350	68
R. P.	1-30 1956	4-12 1955	41	1954	Mod-Adv. Active	LUL + TP 7-Dec-55	0	0	0	0				0	0				2617	106		
R. K.	11-15 1955	12-14 1954	56	1952	Far-Adv. Active	Rul + TP 16-Sept-55	0	0	2/ F	2+	5+	3+	4+	0	0				4220	63.6		
									FLAVOBACILLUS													
H. C.	1-7 1956	5-24 1955	53	Feb. 1955	Mod-Adv. Active	Rul + TP 11-Nov-55	0	0	0	0				0	0				3930	95	2990	43.4
									FLAVOBACILLUS													
H. N.	12-20 1955	8-2 1955	26	1954	Far-Adv. Active	Rul + TP 25-Oct-55	0	0	3/50 F	2+	2+	4+	4+	0	0				4125	109		

TABLE 1 Cont'd

Name	Discharged	Admitted	Age	Onset	Classification of Disease	Operative Procedure	Complications	DEATH	SPUTUM[1]										PULMONARY FUNCTIONS			
									PRE-OP					POST-OP					PRE-OP		POST-OP	
									S	CUL.	CAT	SM	INH	S	CUL.	CAT	SM	INH	VC	MBC	VC	MBC
I. W.		2-17 1955	35	1950	Mod-Adv. Active	Lul + TP 23-Sept-55	0	0	40/ F	4+	+	0	0	0	0				3430	131	2830	114
K. C.		4-1 1955	36	1948	Far-Adv. Active	Rul + TP 4-Nov-55	0	0	3/ F	4+	+	4+	4+	10/ F	4+	0	4+	4+	2560	94	2260	82
H. W.		8-7 1955	24	1949	Far-Adv. Active	SupDivLul+TP 18-Nov-55	Hemo-thorax	0	0	1+	0	0	4+	0	1+	+	4+	4+	2600	105		
F. S.		1-20 1955	43	1943	Far-Adv. Active	Rul + TP 2-Feb-56	0	0	30/ F	4+	2+	4+	4+	0	0				3100	113	1900	78
O. R.		11-4 1955	27	1951	Mod-Adv. Active	Rul+SupSegRII + TP 15-Feb-56	0	0	0	0				0	0				2240	103		
A. G.		7-15 1955	21	1952	Far-Adv. Active	Rul+TP 11-Apr-56	0	0	0	0				0	0				3200	71		
R. C.		11-11 1955	40	1953	Far-Adv. Active	Rul + TP 20-Apr-56	0	0	20/ SOF	1+	+	1+	4+	--	--				4600	100		
A. C.		1-26 1956	24	1955	Mod-Adv. Active	AP-PostSegRul + TP 25-Apr-56	0	0	30/ F	4+	0	4+	4+	--	--				4700	156		
G. S.		8-2 1954	30	1954	Mod-Adv. Active	Rul+ TP 4-Apr-56	0	0	0	2+	+	3+	4+	--	--				1850	126		

A rubber tube is placed in the subcapsular space and is connected to under water drainage. Negative pressure of 4 to 5 cm. of H_2O is used for 24 to 48 hours. This tube is removed on the third to seventh postoperative day.

Case Reports

The first apico-osteoplasty of the type herein described was performed in September, 1955. Since then 17 cases have been completed. The ages ranged from 21 to 53 years. Nine, by the 1955 National Tuberculosis Association standards, were classified as having advanced disease and eight, moderately advanced. Preoperatively 13 cases were sputum positive and resistant to the antimicrobials, while four were sputum negative. Postoperatively 13 became sputum negative whereas four still remained positive on smear and culture. Contralateral tuberculosis involvement, however, was present in the latter four cases. A brief summary of all the cases is to be found in Table I.

As noted postoperative complications were observed in two cases:

The first is H. W., a 24 year old Chinese man who hemorrhaged intrapleurally during the first 24 hours postoperatively. This was attributed to hypofibrinogenemia. He was treated conservatively and a slight degree of captive lung was his only sequela.

Case J. T., concerns a 43 year old white man, who in addition to having bilateral far advanced pulmonary tuberculosis, had severe rheumatoid arthritis and a history of demerol addiction. He had borderline pulmonary function with evidence of marked air trapping. At surgery, cavities in the right upper lobe and superior segment of the lower lobe were removed. In an attempt to achieve maximum space filling the fourth rib was divided in addition to the second and third (this is the only instance of our dividing three instead of two ribs). Postoperatively marked paradoxical breathing

FIGURE 3A FIGURE 3B

Figure 3A: Preoperative x-ray film of patient H. N.—*Figure 3B:* X-ray film of H. N. two weeks after a right upper lobectomy and apico-osteoplasty. The hard and soft portions were brought down to the azygos vein and attached to the mediastinal pleura. The first rib was left in place.

was observed. This along with air trapping, limited respiratory function, and a poor mental attitude prevented the proper expansion of the remaining right lung and no doubt contributed to the resultant empyema and fistula formation.

The following cases are illustrative of those successfully treated with this procedure:

Case H. N., a 26 year old white man was first admitted to National Jewish Hospital on August 2, 1955. Symptoms of tuberculosis began in January, 1954, and the following month a chest x-ray film revealed a lesion in the right upper lung field. He was treated for 13 months with streptomycin, 2 grams weekly and INH, 300 mg. daily. On admission to National Jewish Hospital his sputum was positive for tubercle bacilli on both smear and culture. The organisms were resistant to streptomycin and INH. X-ray film of the chest disclosed a 4 cm. in diameter cavity in the right upper lobe. Bronchoscopy revealed a normal endo-bronchial tree. Pulmonary functions were satisfactory. He was prepared for one month preoperatively with INH 16 mg./kg./day, PZA 40 mg./kg./day and PAS 10 grams daily. On October 25, 1955, right upper lobectomy and concomitant apico-osteoplasty were carried out. The postoperative course was uneventful. The patient was discharged on December 30, 1955, and his sputum remained negative on smear and culture postoperatively. Figures 3A and 3B show the preoperative as compared with the two weeks postoperative x-ray film changes.

Case H. C., a 53 year old white man was first admitted to National Jewish Hospital on May 29, 1955. A routine chest x-ray film in 1949 disclosed what was interpreted as pulmonary tuberculosis inactive. Constitutional signs and symptoms first developed in February, 1955. His sputum was positive on culture and resistant to streptomycin but sensitive to INH. The organisms were atypical and high catalase activity was demonstrated (flavobacillus). X-ray film of the chest revealed an 8 by 5 mm. cavity with adjacent nodose disease in the right upper lung field. On June 16, 1955, he was started on streptomycin 2 grams daily for 28 days and INH 16 mg./kg./day. His sputum became negative on smear and culture on July 22, 1955 and remained negative. Bronchoscopy and pulmonary functions were normal. Because of the rather prominent residual disease in the right upper lobe, resection was carried out. On November, 1955 the right upper lobe was removed and apico-osteoplasty performed. His postoperative course was satisfactory. He was discharged on January 17, 1955 and his sputum remained negative since surgery. Figures 4A and 4B show the x-ray film changes induced by surgery.

Discussion

Since many of the resections for pulmonary tuberculosis performed at National Jewish Hospital at Denver have been done on cases in which

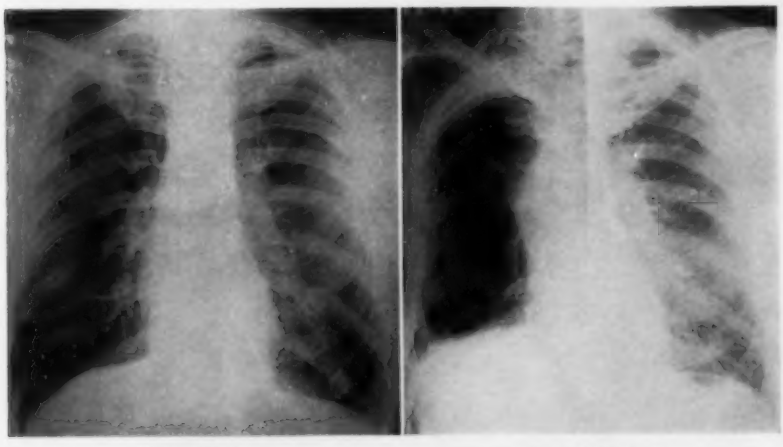

FIGURE 4A FIGURE 4B

Figure 4A: Preoperative x-ray film of patient H. C.—*Figure 4B:* X-ray film of H. C. two weeks postoperative, following a right upper lobectomy and osteoplasty. The first rib was left in place. Apicopexy was carried down to the azygos vein.

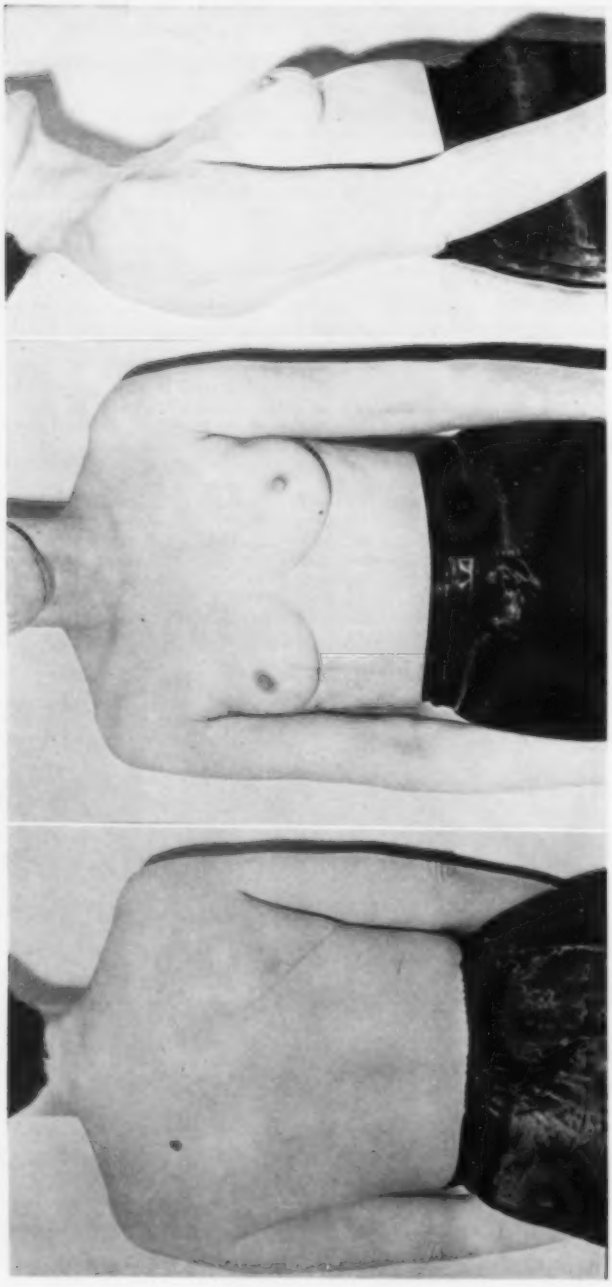

FIGURE 5A FIGURE 5B FIGURE 5C

Figures 5A, 5B, and 5C: Front, back, and profile views demonstrating the cosmetic results in a patient who had a right upper lobe resection and concomitant apico-osteoplasty.

the remaining lung tissues demonstated an inability to expand, space reducing procedures have frequently been necessary.

In our experience the apico-osteoplasty have proved to be an excellent procedure. Unlike the classical thoracoplasty with apicolysis this procedure does not create any significant additional trauma or blood loss; nor is it accompanied by the incidence of infections, fistula formation or the other hazards associated with extraperiosteal plombage.

Although the Bjork thoracoplasty[3] in our experience has been a satisfactory space filling procedure it has tended to lengthen the operating time and to produce additional trauma and blood loss. The space of Semb[2] became infected in two instances.

Our experience with "architectural reconstruction of the lung" as recently advocated by French authors[8] has proved disappointing in the few cases in which it was utilized. We feel that a procedure requiring compensatory emphysema of the ipsilateral and contralateral lung, in tissue that has previously or is presently diseased with tuberculosis, is not without hazard.

With the technique presented it has been found that the space remaining after the upper lobectomy with or without resection of the superior segment of the lower lobe can be handled adequately. The level of the new apex can be as low or lower than the aortic arch on the left and the azygos vein on the right. If there is little or no apical pleura remaining because of necessary extrapleural dissection in the upper lobectomy, the endothoracic fascia alone can be imbricated.

The imbrication and fixation of the soft portion immobilizes the bony plate in an efficient manner. Consequently, paradoxical breathing is kept to a minimum and easily controlled with a shot bag or compressive dressing. Care is taken in removing the small segments of the ribs subperiosteally in order to maintain the integrity of the soft tissue of the plate.

Likewise, only enough bone is removed from the 2nd and 3rd ribs to provide for free overriding of the cut ends permitting a satisfactory descent of the hard plate.

The time required for this procedure is from 30 to 45 minutes; the blood loss is minimal and usually does not exceed 200 to 300 cc. The cosmetic result is excellent with no noticeable disfigurement. (Figures 5A; 5B; 5C).

The resultant subscapular space is moderately large but has been effectively handled by a tube connected to the under water drainage and negative pressure. Generally about 400 cc. of serosanguineous fluid drains from the area in the first 24 hours and thereafter the drainage is slight.

SUMMARY

Seventeen cases in which the apico-osteoplasty was performed have been presented. Postoperative morbidity was observed in two. Four prior to surgery had negative sputa and no postoperative change was observed. Nine had conversion of sputum to negativity following the

procedure. Of the four who remained positive all had contralateral disease.

The apico-osteopexy has been proved to be a satisfactory space filling procedure following resection in advanced pulmonary tuberculosis. The advantages are: (1) Its adaptability to varying amounts of lung resected; (2) Minimal trauma and blood loss; (3) Avoidance of multiple stage procedures and foreign body plombage; (4) Minimal paradoxical motion; (5) Absence of cervicothoracic spine deformity.

RESUMEN

Se presentan 17 casos en los que se ha practicado apico-osteoplastía. Hubo morbilidad postoperatoria en dos. En cuatro había esputos negativos antes de la operación y no se observó cambio postoperatorio. Nueve tuvieron conversión haciéndose negativos de esputos después de la operación. De los cuatro que siguieron siendo positivos todos tenían enfermedad del pulmón opuesto.

La apico-osteopexia se ha mostrado ser un procedimiento satisfactorio para llenar el espacio residual después de resección en tuberculosis avanzada. Las ventajas son: (1) Su adaptación a volúmenes diversos de pulmón resecado; (2) trauma mínimo y poca pérdida de sangre; (3) el evitar procedimientos en tiempos múltiples y el uso de cuerpos extraños para plombaje; (4) mínimo de movimiento paradójico; (5) ausencia de deformación cérvico-torácica.

ZUSAMMENFASSUNG

Bericht über 17 Fälle, bei denen die Apico-Osteoplastik ausgeführt wurde. Postoperative Morbidität wurde in 2 Fällen beobachtet. 4 hatten vor dem Eingriff ein negatives Sputum und postoperativ wurde eine Änderung nicht beobachtet. Bei 9 wurde das Sputum negativ im Anschluss an den Eingriff. Von den 4, die positiv blieben, hatten alle kontralaterale Prozesse.

Die Apico-Osteoplastik hat sich als ein befriedigendes, den Hohlraum im Gefolge einer Resektion bei fortschreitender Lungentuberkulose füllendes Verfahren erwiesen. Die Vorteile sind: (1) Ihre Anpassungsfähigkeit an verschiedene Grade von Lungenresektionen; (2) Ein minimales Trauma und Blutverlust; (3) Vermeidung von Methoden mit vielfachen Sitzungen und Fremdkörperplombierung; (4) Minimale paradoxe Beweglichkeit; (5) Ausbleiben von Deformierung der Cerviko-thorakalen Wirbelsäule.

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Hemorrhage into Thymic Tumors: Produced by Rapid Deceleration and Trauma

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Tumors and cysts of the thymus are no longer regarded as rare lesions. They are detected on routine x-ray film examination of asymptomatic individuals, those complaining of cough, chest pain, or dyspnea, or in patients with myasthenia or superior venal caval syndrome. Trauma, producing acute hemorrhage into a thymic cyst or tumor, initiating severe precordial pain as the only complaint, has not been specifically recorded.

Case 1: A 21 year old white man suffered cerebral concussion, rupture of the spleen, multiple lower left rib fractures, multiple lacerations of the face and extremities, and contusion of the left kidney in an automobile accident on December 30, 1955. Splenectomy was performed the same day. Three days later he was air evacuated to Parks Air Force Base Hospital. Hematuria persisted for several days, after which the urine became clear. At the end of one week of hospitalization the left lower chest, abdominal, and leg pain subsided but was replaced by steadily increasing sharp precordial pain on inspiration, localized to the third and fourth interspace to the left of the sternum. On January 7, an x-ray film of the chest revealed an abnormal shadow along the left border of the heart (Figure 1). The most recent film taken prior to the accident was obtained which was normal. Angiocardiography was performed on February 6, 1956 but visualized normal mediastinal great vessels. Contrast medium did not enter the mass. The left hemithorax was explored through an anterior incision on February 21,

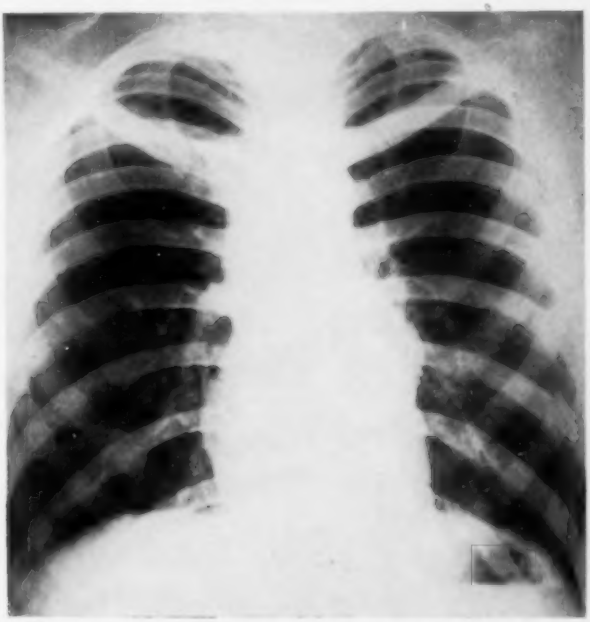

FIGURE 1 (Case 1): Posterio anterior film of chest January 7, 1956 showing an abnormal shadow along the left border of the heart.

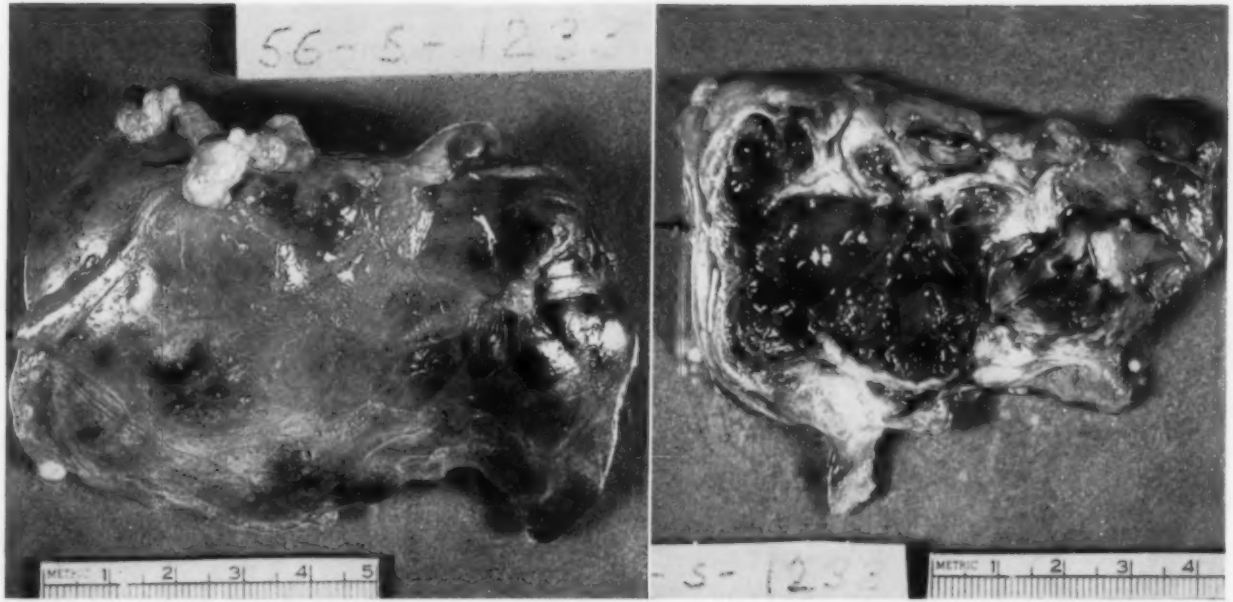

FIGURE 2

FIGURE 3

Figure 2 (Case 1): Unopened thymic cyst 3.5 x 4.5 x 8 cm. in size. Note distension.—*Figure 3* (Case 1): Opened thymic cyst. Note presence of the organized blood clots attached to the cyst wall.

FIGURE 4

FIGURE 5

FIGURE 6

Figure 4 (Case 2): Posterior anterior film of chest July 12, 1955 showing an 8 cm. tumor mass in the right chest.—*Figure 5* (Case 2): Right lateral x-ray film showing anterior position of the thymic tumor.—*Figure 6* (Case 2): Note the undifferentiated cellular pattern of the tumor and the presence of large quantities of extravasated blood.

1956, preparations having been made to transect the sternum and treat a traumatic aneurysm should it be encountered. Instead a 3.5 x 4.5 x 8 cm. thymic cyst distended and lobulated, arising from the left inferior horn of the thymus, was found and removed (Fig. 2). The cyst contained masses of clotted blood and the cyst wall contained elements of thymic tissue (Fig. 3). He made an uneventful recovery and has since returned to duty.

It is presumed that at the initial trauma, bleeding into a small thymic cyst occurred. This produced a situation similar to a subdural hematoma into which serous fluid moved by osmosis to produce the severe distension of the cyst found at thoracotomy.

Case 2: A 23 year old white officer, a jet pilot, was completely well until July 1, 1955 when he performed a power dive in a jet plane. Shortly after landing he was seized with excruciating substernal pain radiating through the chest to the back, requiring large doses of demerol for relief. On the following day an x-ray film revealed an 8 cm. spherical mass arising from the mediastinum and projecting into the right chest (Figs. 4 and 5). He was referred to Parks Air Force Base Hospital on July 12, 1955. Except for dullness to percussion to the right of the sternal border between ribs three and six, physical examination was negative. All laboratory tests, bronchoscopy, and sputum examinations were negative.

On July 18, 1955 right exploratory thoracotomy was performed. The tumor mass was found to arise from the inferior horn of the right thymus and had extensively infiltrated the base of the heart and great vessels. There was gross hemorrhage visible in the periphery of the tumor mass. Frozen tissue examination of a biopsy disclosed an undifferentiated carcinoma of the thymus with diffuse hemorrhage, confirmed on final tissue examination (Fig. 8). The chest was closed without attempting further resection. The patient recovered from the thoracotomy. In September 1955 a re-exploration was done elsewhere. The lesion was again found to be inoperable and the patient expired 36 hours after surgery.

This thymic carcinoma, despite its large size, was completely asymptomatic until a severe trauma was inflicted. A pilot, pulling out of a power dive, is subjected to many time the force of gravity and the pressure is especially concentrated across the chest. Moreover, the thoracic contents being more mobile than the bony thorax will "whip" forward during deceleration. It is difficult to assess how much of the size of this tumor was due to blood, but the impression grossly was that almost half the bulk of the tumor was composed of blood.

Discussion

This paper does not attempt to review the world literature on a most complex subject. There is no record of massive hemorrhage into a solid thymic tumor in recent communications.[1-8] Thymic cysts whose contents are bloody or chocolate in color have been described.[2, 5, 6] Recently two thymic cysts producing chest pain and containing bloody fluid have been reported.[2] It is not reported how sudden or severe the precordial pain was in any of these cases. In this day of tremendous speed and severe acceleration and deceleration, it is not surprising that such hemorrhage would occur. The thymus, situated just beneath the rigid plate of the sternum, is ideally located to receive a sharp blow as sudden deceleration occurs.

CONCLUSION

Two cases, previously asymptomatic, suffered severe precordial pain following rapid deceleration. Hemorrhage into a thymic carcinoma in one instance and into a thymic cyst in the other is presumed to have initiated the pain.

References will appear in reprints.

Excision of Massive Ganglioneuroma of Mediastinum

A Case Report

CHARLES H. LASLEY, M.D.*
Clearwater, Florida

Benign tumors of the mediastinum may on occasion reach massive proportions. Previous reports on mediastinal tumors made by Clagett and others include a fibroma weighing over 4000 gm successfully removed, and a lipoma (autopsy specimen) of 12,000 gm.

Ganglioneuroma of the mediastinum is a rare lesion and is almost always posterior in origin. Three of 71 cases reviewed by Kent and Blades were in the anterior mediastinum.

Case Report

L. R., Gorgas Hospital No. 662967, 41 year old Salvadorean blacksmith, was admitted November 24, 1952 with a diagnosis of "asymptomatic calcified mass left chest." This large mass was first noted in the left hemithorax by x-ray film in 1948, with enlargement in November 1952 (Figures 1 and 2). There was no history of traumatic or inflammatory episode involving the chest. Physical examination revealed only dullness with absent breath sounds in the left lower chest with displacement of the heart and trachea to the right.

Left lateral thoracotomy was performed December 4, 1952 with resection of the sixth rib. On opening the pleura the massive size of the tumor prevented free access to the remainder of the pleural cavity and at this time his blood pressure dropped to 70/50

FIGURE 1

FIGURE 2

Figure 1: Preoperative posteroanterior x-ray film showing mediastinal displacement.—*Figure 2:* Lateral x-ray film showing size and calcification of tumor.

*Formerly Chief of General Surgery Section, Gorgas Hospital, Ancon, Canal Zone.

due to interference with cardio-respiratory function by the weight of the tumor. Lateral (outward) traction on the tumor mass improved cardiac function and the blood pressure rose to 90/60 while traction was maintained during subsequent dissection. The fifth and seventh ribs were also divided to allow removal of the tumor.

The mass appeared to arise from the pericardium with anterior displacement of the left phrenic nerve. A 3 x 4 cm. portion of pericardium was excised with the tumor. There were only loose areolar and small vascular attachments to the mediastinal pleura.

His hypotension persisted for one week but he had an otherwise uneventful post-operative course. At follow-up four years later he was asymptomatic with no evidence of recurrence.

Grossly the tumor was a smooth pink firm lobulated mass measuring 20 x 20 x 28 cm. and weighed 3050 gm. It was densely calcified and had to be cut with a bone saw. Microscopically the soft tissue showed whorls of fibrous connective tissue containing large distorted ganglion cells, and Bodian stain demonstrated neurites at the periphery. The tumor was coded by the AFIP as a sclerosing ganglioneuroma of the mediastinum.

Dermoid Cyst of the Mediastinum Communicating with a Bronchus

Report of Two Cases

KIYOSHI INADA, M.D., F.C.C.P.*

Okayama City, Japan

Benign mediastinal tumors as a group is a relatively uncommon disease. Recently, however, as a result of the routine use of x-ray films in medical examinations and mass surveys, symptomless cases of mediastinal tumors are being found with increasing frequency.

Dermoid cysts and teratomas are two of the most common tumors in the mediastinum. Though inherently benign, they are not uncommonly associated with various complications such as infection, perforation, and degenerative change to malignancy. In such cases, it is often difficult to arrive at a correct diagnosis before pathognomonic signs appear.

Recently, we have had two cases of dermoid cysts of the mediastinum communicating with a bronchus presenting interesting features from the diagnostic standpoint. It is our purpose to report these cases and draw attention to the complications associated with mediastinal dermoid cysts, because relatively few reports discussing the subject are found in the literature.

Case Reports

Case 1: A 30 year old man was admitted to the hospital on September 2, 1953, with the chief complaints of chest pain, low grade fever, and fragments of hair in his sputum. The history of the present illness dates back about 10 years when he developed pain in the left chest accompanied by fever. This was diagnosed as tuberculous pleurisy and was so treated. Because of persistent low grade fever, he sought admission to the University Hospital two months later, where he was admitted with an admission diagnosis of tuberculous lymphadenitis of the left hilum and was kept in bed for three

*Instructor in Surgery, Okayama University Medical School.

months. During this time, he had slight cough which was productive of scanty sputum, and a feeling that he was not completely well. His physical activities were restricted for fear of an exacerbation of the illness. During all this time he also had several attacks of upper respiratory infection and on one occasion had blood-tinged sputum. He also recalled that six months prior to admission, he had fever and bilateral chest pain, which was diagnosed as pneumonia and was treated with penicillin. At that time he coughed up white hairs and sebaceous matter whenever he expectorated. A diagnosis of mediastinal dermoid cyst was made and he was referred to the surgical clinic.

Physical examination revealed a well developed and well nourished man who did not appear chronically ill. Breath sounds were slightly diminished and few moist rales audible over the anterior part of the left chest. Other signs were essentially negative. There was no remarkable finding on laboratory examinations.

Chest roentgenograms revealed a triangular area of consolidation in the left hilar region protruding into the left lung field (Fig. 1). The lateral film demonstrated a definite shadow simulating a tumor the size of a hen's egg behind the sternum (Fig. 2). No abnormality was found in the lungs or bronchi on planigrams.

Anterior mediastinotomy was done under local anesthesia on September 4, 1953. After resecting small segments of the second, third and fourth ribs, a cystic tumor was found between the pericardium and the upper lobe of the left lung. It was firmly adherent to the lung and on dissection, a small communication was found between the cyst and the anterior segmental bronchus of the left upper lobe. The anterior segment was atelectatic. The adhesions were sharply dissected and the tumor was removed without undue difficulty. Lying close and beside the cyst was a smaller tumor about the size of the tip of an adult thumb, and which was thought at first to be a daughter tumor. Further dissection however, revealed that it was a lymph node which had developed in its entirety into an abscess.

Pathological examination of the resected specimen revealed a cyst 3.5 x 5.0 x 3.0 (cm.) in size, weighing 388 gm., which on section was found to contain long white hairs and yellowish muco-purulent and sebaceous matter. Microscopical sections showed a typical picture of dermoid cyst.

Case 2: A 10 year old boy was admitted to the hospital with the chief complaint of abundant foul sputum of six months duration. The history revealed that he had several episodes of upper respiratory infection since the age of five when he developed pneumonia. A suspicion of dextrocardia was also entertained after an x-ray film examination of the chest at that time (Fig. 3). Two years prior to the present

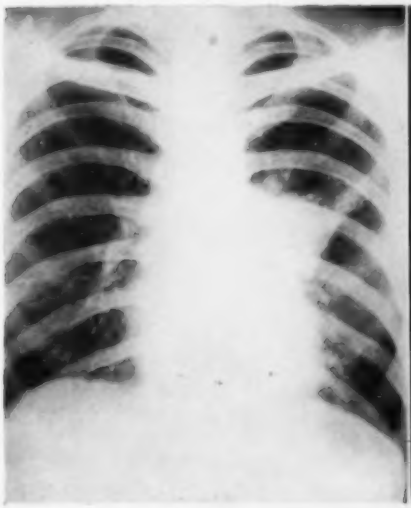

FIGURE 1

FIGURE 2

Figure 1 (Case 1): Posteroanterior chest roentgenogram.—*Figure 2* (Case 1): Left lateral roentgenogram showing tumor-like shadow behind the sternum.

admission, he developed abundant sputum which did not respond to several antibiotics. He also developed high fever when the amount of sputum decreased. Six months before he sought admission his sputum became fetid. He also noted that he was more comfortable sleeping in the left lateral position because of freedom of attacks of severe coughing. Physical examination revealed a thin anemic boy who appeared chronically ill. Examination of the chest showed it to be symmetrical with dullness to percussion and diminished breath sounds over a narrow area in the third and fourth intercostal spaces along the right sternal border.

Chest roentgenograms on admission showed a sharply defined round shadow in the right mid and lower lung fields, simulating an abscess or cyst containing air and fluid. This was continuous with the heart shadow (Fig. 4). The lateral film showed this condition was located in the middle lobe (Fig. 5). Bronchography was done under general

FIG. 3

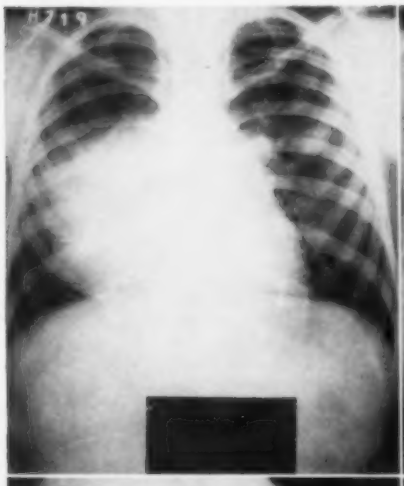

FIG. 4

FIG. 5

FIG. 6

Figure 3 (Case 2): Posteroanterior chest roentgenogram taken at the age of five.—*Figure 4* (Case 2): Posteroanterior chest roentgenogram on admission.—*Figure 5* (Case 2): Right lateral roentgenogram.—*Figure 6* (Case 2): Right lateral bronchogram showing middle lobe bronchus displaced posteriorly and inferiorly. Note markedly decreased fluid in the cyst after intratracheal aspiration before bronchography.

anesthesia. After over 100 cc. of sputum was aspirated with the patient in the right lateral position, the contrast medium was instilled through a tracheal catheter. A good bronchogram was obtained but the contrast medium had not entered the abscess cavity. The middle lobe bronchus was found displaced posteriorly and inferiorly, otherwise no marked change in the bronchial tree was noted (Fig. 6). Fluoroscopic inspection revealed good function of the right diaphragm. Examination of a chest roentgenogram taken at the age of five years showed a sharply defined tumor-like consolidation occupying most of the hemithorax and connected to the heart shadow (Fig. 3). Sputum which amounted from 150 cc. to 200 cc. daily, failed to reveal hair or tubercle bacilli. Culture of the sputum showed Candida albicans and Gram-positive diplococci.

Right thoracotomy was done under Pentothal-Cyclopropane-Ether endotracheal anesthesia on Aug. 30, 1956. The right pleural cavity was entered through the anterolateral approach and diffuse loose adhesions were encountered between the pleura and the entire right lung. A round cystic tumor was found in the anterior mediastinum, partly covered by the middle and lower lobes, and adherent to both lobes, to the pericardium and to the diaphragm. During the dissection, a communication between the cyst and a branch of the medial segmental bronchus of the middle lobe (B_5) was found. The tumor was extirpated without difficulty.

Pathological examination of the resected specimen showed a cyst, 5.5 x 5.0 x 5.5 cm. containing muco-purulent material; no hair was found. Thickness of its wall ranged from 2 mm. to 5 mm. Microscopic sections showed a typical picture of dermoid cyst.

Discussion

As complications of dermoid cysts of the mediastinum, the following have been mentioned: simple mechanical compression of vital structures, development of infection, adhesions and subsequent rupture into the surrounding tissues, especially into bronchi, and malignant changes. Infection of a cyst is probably secondary to inflammation of the surrounding tissues, especially the lungs. Infection through the blood stream is also probable. Following infection of the cyst, adhesions develop between it and the surrounding lung tissue, with subsequent rupture of the cyst into a bronchus occurring sooner or later.

Rupture of a cyst is associated with the sudden onset of acute symptoms such as fever, severe and distressing cough, hemoptysis, and coughing up of hair or sebaceous matter, the last being the only pathognomonic symptom for an intrathoracic dermoid cyst that has ruptured into a bronchus. In some cases, a cyst-bronchial communication may develop and remain silent. Such communications can exist for years without causing serious symptoms. There are relatively few reports with special reference to the incidence of this kind of complication in mediastinal dermoid cysts. Rusby[1] found 23 cases (11 per cent) where specific mention was made of trichophytosis among 245 cases of mediastinal dermoid cysts and teratomata collected from the world literature up to 1939. Laipply[2] gave a figure of 33 (13 per cent), among 245 cases of cysts and cystic tumors of the mediastinum collected from the literature. However, in his series, the true incidence should be considered to be 22 per cent (33 out of 154 cases) excluding the various kinds of cysts that do not contain hair originally and other than dermoids and teratomata.

The author[3] was able to collect up to date from the Japanese literature 76 cases of dermoid cysts and teratomata of the mediastinum. This number is rather small compared to the figures cited by the two authors mentioned above. However, the author believes that it would not be too impertinent to attempt to draw some discussion on the basis of these statistics. A cyst-bronchial communication was considered existent in 21 cases. Tricho-

phytosis was noted in 19 of the 21. Including the two cases presented in this report, the incidence of cyst-bronchial communication in mediastinal dermoid cysts is 27 per cent (21 of 78 cases). This figure is rather high and is significant in that the incidence of this complication is more than twice that of the figure in Rusby's series.

Twenty-seven of a total of 78 cases in the author's series were teratomas. Five of the 21 cases where a cyst-bronchial communication developed were also teratomas. The rest were dermoid cysts. It should be noted that this complication is more common in dermoid cysts than in teratomas. This is easily understood if we consider the fact that teratomas do not always have cystic structures. It is important enough to merit recognition that this complication developed under the age of 26 years in 13 of the 21 cases in the author's series.

In the first case it could not be determined when infection of the cyst developed because no chest x-ray film was available before he noticed trichophytosis.

It is interesting also that in the first case a lymph node that has entirely degenerated into an abscess was present beside the cyst. This node may be considered to be the source of infection of the cyst. It should also be emphasized that the size, shape and configuration of the shadow of a tumor on the x-ray film may be deformed or distorted with the presence of superimposed infection.

In the second case, infection and rupture of the cyst probably developed at the age of five years when the patient had pneumonia-like symptoms. No marked change was noticeable in the subsequent serial x-ray films of the chest, but it was evident that the tumor shadow in the first film distinctly decreased in size in the succeeding films.

Dermoid cysts of the mediastinum should not be considered benign from the standpoint of complications as mentioned above. Surgical removal should be done as early as possible after detection.

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Teaching Chest Disease
Thinking vs. Believing

THEODORE H. NOEHREN, M.D., F.C.C.P.*
Buffalo, New York

There is a tremendous paradox in medical education today; we teach things we *think* are true but actually do not *believe*. Perhaps it is not such a characteristic of medical education as of our whole profession, or even society. The "do as I say, not as I do" school of individuals is well-known in almost all spheres of activity. However, in medicine, especially medical education, it has rather serious implications.

In chest disease we teach, for example, that the annual routine chest roentgenogram is a fundamental necessity. However, a casual poll among our thoracic confreres reveals a surprisingly small number who have followed this principle unless they are old "lungers," which puts their practice into another category. We teach Physical Diagnosis of the chest with a careful review of all the classical terms and procedures. Yet, on rounds and in our chest conferences, the arena of chest disease, such terms are rarely ever heard, much less the details of a carefully patterned physical examination of the chest. The reason is obvious. The students follow our examples at the bedside, for the carefully taught theoretical routines are not always carefully practiced. The call for the ready roentgenogram is considerably more routine.

Either we believe in these practices or we do not. There is no doubt we think they are valuable or we would discontinue teaching them. What it amounts to is that we do not really *believe* enough in what we *think* is important, excuses of insufficient time etc., to the contrary.

Another area in which we are paradoxical, though perhaps not so much as in some of the above, is in history taking. We teach and write quite routinely that an adequate history is the cornerstone, perhaps as much as 90 per cent of the accurate diagnosis. Not only is the taking of the history important, but the recording of it is equally so. We think so, but a survey of routine records, particularly office records, does not generally reveal that we believe it practical enough to use.

A final and more desperate demonstration of the thinking vs. believing paradox among us is the behavior when we are the victims of our own special disease entities. Administrators of tuberculosis sanitoria can tell endless stories of physician patients. Surgery for an undefined pulmonary lesion may be mandatory for our patient, but, when it comes to the lesion in ourselves the story is often quite different, at least delayed. Many of us trust our confrere with the care of any lesion but our own. We implicitly

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The second in a new series of articles prepared under the sponsorship of the Council on Undergraduate Education of the American College of Chest Physicians.

trust many laboratory and therapeutic measures in anyone but ourselves. We tell the patient not to worry when, as patients, the physician is a notable exception. In these and many areas we think (quite honestly) a thing is of value but not quite enough to accept it emotionally.

This is nothing more than "human nature" perhaps, and could be dismissed as such if it did not profoundly influence our teaching effectiveness. It is folly to fool ourselves into the belief that students are not keenly aware of this paradox. Perhaps they do not have the opportunity to see all these aspects but many more are apparent than we realize.

If we really believe that a proper history and physical examination are fundamental, the best way for us to teach it is to practice it. If we believe preventive measures in tuberculosis involve routine roentgenograms and skin testing we should practice it on ourselves, on the students and consistently in our patients. If we really think physical findings are so important we should include them in case conferences before bowing to the roentgenologist's expert opinion . . .

The question arises whether we are teaching for examinations or for practice. There is a general undercurrent of student opinion that as soon as they can get over all this formal (insincere?) training they can get down to the everyday practice of medicine, they can write histories the way they want to, and not go through all the "unnecessary" maneuvers. Either we must convincingly demonstrate the necessity of what we now teach or stop teaching it. We should recognize that we may be teaching a fantasy of practice, good though we may think it is. Perhaps it is a combination of improving our own practice and improving on our approach to students with practiced principles. When curricula are as crowded as they are today it becomes even more important to winnow our teaching into what we actually believe.

The old aphorism "Them what can, does; them what can't, teaches" may not be so far from stating the paradox of thinking and believing. The effective teacher is the one who stimulates the student with enthusiasm for emulating the methods he has seen work well.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

One of the most successful international medical congresses was held in Washington, D. C., May 5-10, under the capable direction of Dr. Paul H. Holinger, Chicago, a Fellow of the American College of Chest Physicians, and Secretary-General of the Congress. The congress was acclaimed as the best organized international medical meeting sponsored by this society. Speaking for the English guests, Sir Victor Negus, a delegate from England, said, "This Sixth International Congress of Otolaryngology will be a challenge to the organizers of future congresses and will be extremely difficult to duplicate." This is a fine tribute to the efforts of Dr. Holinger and his committee who labored untiringly to make certain that every detail of organization was carried out to perfection.

The congress was attended by more than 2,000 physicians and their families from 48 countries. There were simultaneous translations in German, French, Spanish, and English in the main assembly hall. The motion picture sessions were the highlights of the congress and the hall was filled to capacity at each showing.

Vice-President Richard M. Nixon was the principal speaker at the convocation in Constitution Hall on Sunday night, May 5. On Tuesday night, the National Symphony Orchestra and the Howard University choral group gave a splendid concert in Constitution Hall.

Special tours of the District of Columbia and other social activities were planned to give delegates from other countries an opportunity to learn something of our capital and customs. A hospitality lounge at the Statler Hotel, sponsored by [illegible]derle Laboratories, furnished refreshments throughout the meeting.

The closing banquet on Thursday night, May 9, was the climax of this outstanding congress. Dr. Arthur W. Proetz, St. Louis, President of the congress, presided at the banquet and introduced the guests of honor. The congress was attended by many Fellows of the American College of Chest Physicians throughout the world who specialize in bronchoesophagology.

SIXTH CONGRESS OF THE INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY

The Sixth Congress of the International Bronchoesophagological Society was held in Philadelphia, May 11-13, under the presidency of Dr. Theodor Hunermann, Dusseldorf, Germany. Arrangements for the congress were directed by Dr. Chevalier L. Jackson, Philadelphia, Executive Secretary of the society, and his competent staff of assistants.

The congress opened Saturday night, May 11, with a reception at the Bellevue Stratford Hotel and the first scientific session was presented Sunday morning, May 12. Luncheon was served at the Philadelphia Country Club, and after a tour of the beautiful Jardins de Banlieue, a lovely buffet supper was served at the Hotel Warwick. The second scientific session was presented on Monday morning, May 13, followed by a visit to the clinics of otolaryngology and bronchoesophagology in Philadelphia.

The ladies were entertained at a fashion show sponsored by Bonwit-Teller at the Bellevue Stratford Hotel and both the physicians and their wives were given a reception that afternoon at the Philadelphia Museum of Art. The congress closed with a banquet at the Bellevue Stratford Hotel sponsored by the Otolaryngology Society of Philadelphia.

Dr. Jackson and his assistants are to be congratulated upon the splendid arrangements for this congress which was attended by more than 250 physicians and their wives from various parts of the world. Many of the bronchoesophagologists will be attending the 23rd Annual Meeting of the American College of Chest Physicians to be held at the Hotel Commodore, New York City, May 29-June 2.

College Chapter News

CUBAN CHAPTER

A postgraduate course on clinical pulmonary physiology and respiratory function testing, sponsored by the Cuban Chapter of the College, was given at the Curie Hospital, Havana, March 18-23, 1957, under the direction of Drs. Frank Barrera, Robert Douglas and Gabriel G. Regalado.

Left to right: Drs. Rodrigo Bustamante, Secretary-Treasurer, Cuban Chapter; F. J. Menendez, College Governor for Cuba; A. Nuñez, President, Cuban Chapter; G. G. Regalado, F. Barrera and Roberto Douglas, lecturers; J. Llambes, Director, Curie Hospital; A. Navarrete, College Regent for Cuba; and Luis Romaguera, Vice-President, Cuban Chapter.

SOUTHERN CHAPTER OF SOUTH AFRICA

The 9th annual meeting of the Southern Chapter of South Africa was held in Cape Town on April 1. The following officers were re-elected:

President:	David P. Marais, Cape Town
Vice-President:	Theodore Schrire, Cape Town
Secretary:	Hendrik Muller, Cape Town
Treasurer:	Michael Bailey, Cape Town

Plans for future meetings were discussed and arrangements made for scientific sessions to be held with local meetings of the Medical Association of South Africa. At the next clinical meeting, Dr. Thomas J. Dry who has recently returned to South Africa after 25 years at the Mayo Clinic, Rochester, Minnesota, will lecture on "What Can Be Done Today for Acquired and Congenital Heart Disease."

Following the business meeting, the 87th clinical session of the chapter was held, at which Dr. David P. Marais, President, presided.

LOUISIANA CHAPTER

The Louisiana chapter held its spring meeting at the Veterans Administration Hospital, New Orleans, on April 5. The following program was presented:

"Open Healing of Tuberculous Cavities"
Oscar Auerbach, East Orange, New Jersey

"Pulmonary Vascular Patterns in Disease"
Coleman B. Rabin, New York City

MIDDLE EAST CHAPTER

The Third Annual Congress of the Middle East Chapter will be held August 9-11 at the Hotel Ambassador, Bhamdoun, Lebanon. The following subjects will be discussed:

Home Treatment of Tuberculosis

Bronchopulmonary Carcinoma

One section of the program will be devoted to papers on other aspects of diseases of the chest, and scientific films will be shown. Those wishing information concerning this congress may write to Dr. Wagih K. Sabbagh, Post Office Box 3847, Beirut, Lebanon. All physicians are invited to attend.

HONG KONG AND CHINA CHAPTER

The Hong Kong and China Chapter met jointly with the Hong Kong Branch of the British Institute of Radiology at the Hong Kong Sanatorium and Hospital on April 25. Mr. Van Bars of Delft, Holland, spoke on the Odelca camera method of radiography, illustrating his lecture with a film on the subject and another on mass chest radiography. Drs. Mary Aquinas and Alan Chan presented chest films and Dr. C. H. Tsao showed a film on the thoracic bones. The program was followed by a discussion of the possible dangers from frequent irradiation of patients by diagnostic x-ray examination, especially of the chest.

NEW CHAPTER OFFICERS

ALABAMA CHAPTER

President:	Kellie N. Joseph, Birmingham
Vice-President:	Arthur Calix, Decatur
Secretary-Treasurer:	Justus M. Barnes, Montgomery

ARIZONA CHAPTER

President:	D. W. Melick, Phoenix
Vice-President:	William B. Steen, Tucson
Secretary-Treasurer:	B. L. Snyder, Phoenix (re-elected)

GEORGIA CHAPTER

President:	F. Levering Neely, Atlanta
Vice-President:	James L. Alexander, Savannah
Secretary-Treasurer:	M. Bedford Davis, Jr., Atlanta

OKLAHOMA CHAPTER

President:	Richard M. Burke, Oklahoma City
Vice-President:	Robert M. Shepard, Jr., Tulsa
Secretary-Treasurer:	George L. Winn, Oklahoma City (re-elected)

TENNESSEE CHAPTER

President:	Leland M. Johnston, Jackson
Vice-President:	J. J. Callaway, Nashville
Secretary-Treasurer:	W. W. Hubbard, Nashville

WISCONSIN CHAPTER

President:	George A. Hellmuth, Milwaukee
Vice-President:	Ross C. Kory, Wood
Secretary-Treasurer:	Leon H. Hirsh, Milwaukee (re-elected)

BED REST IN PULMONARY TUBERCULOSIS

Report of the Sub-committee on Bed Rest in Pulmonary Tuberculosis

There has been some moderate relaxation in the strictness of the application of bed rest in the treatment of pulmonary tuberculosis since the advent of anti-tuberculosis chemotherapy, but it is generally considered as a basic requirement of therapy. This is true in spite of deliberate programs in some areas to de-emphasize the importance of bed rest for the purpose of investigation.

In discussions of the treatment of tuberculosis, the intense interest in methods of use of the various drugs has occupied the attention of the phthisiologist to such a degree that little has been said about the need of rest as a basic part of therapy. To the man in active practice of chest diseases, this need is generally understood and accepted, but to the person who casually reviews the literature, it is easy to gain an erroneous impression regarding the over-all program of tuberculosis treatment. Since an increasing amount of tuberculosis is being cared for by those not specially trained in this disease, the lack of information regarding the whole treatment picture can lead to disastrous results. It is the feeling of this committee that a strong statement should be made reaffirming the importance of bed rest and its proved value in the regimen of tuberculosis treatment and that this statement should receive the widest possible attention. It is believed that bed rest should be maintained until the lesion is stabilized, as indicated by bacteriologic, x-ray, and clinical evidence.

This committee also wishes to reaffirm the desirability of at least starting the patient's care under sanatorium conditions for the purpose of patient indoctrination, the evaluation of his clinical problem, and the initiation of the various therapies.

Buford H. Wardrip, M.D., Chairman

Thomas G. Heaton, M.D.
Leon H. Hirsh, M.D.
Daniel Jackson, M.D.
John S. Packard, M.D.
Harry Shubin, M.D.
Giles H. Wolverton, M.D.

NEWS NOTES

Dr. Frederic W. Holcomb, Kingston, New York, was elected to the Board of Trustees of the Medical Society of the State of New York.

Dr. Orval R. Withers, Kansas City, Missouri and **Dr. Merle W. Moore,** Portland, Oregon, have been named President and President-Elect respectively of the American College of Allergists.

Dr. Hawley H. Seiler, Tampa, Florida, presented the paper "The Surgical Treatment of Pectus Excavatum" at the 25th Annual Assembly of the Southeastern Surgical Congress held in St. Petersburg recently.

Dr. William B. Bean, Iowa City, Iowa, served as a clinical leader of the closed circuit television program "Grand Rounds #5" of the University of Illinois College of Medicine and The Upjohn Company.

AMERICAN HEART ASSOCIATION RESEARCH APPLICATIONS

Applications by research investigators for support of projects to be developed during the fiscal year beginning July 1, 1958 are now being accepted by the American Heart Association. The deadline for research fellowship applications and established investigatorships is September 15, 1957. Applications for grants-in-aid must be made by November 1, 1957. Address inquiries to the American Heart Association, 44 East 23rd Street, New York 10, New York.

Obituary

EVARTS A. GRAHAM

1883-1957

Dr. Evarts A. Graham, dean of American surgeons, died March 4, 1957 of lung cancer. Dr. Graham was born in Chicago, the son of the late Dr. David Wilson Graham and Mrs. Ida Barned Graham. He received his M.D. degree from Rush Medical College in 1907. He practiced surgery in Mason City, Iowa. Prior to World War I he joined the Army Medical Corps, becoming a Major in charge of a hospital in France. It was during his Army Service that he pointed out the danger of too early operation of the pleural cavity of empyema, and as a result, the mortality of this serious complication dropped precipitously. In 1919, he became Professor of Surgery at Washington University School of Medicine and Chief Surgeon at Barnes Hospital until he retired in 1951.

In the 1920's, his knowledge of chemistry served him well, when he introduced the technique of cholecystography, which revolutionized the diagnosis of gall bladder disease. Following this work, he devoted himself to the study of diseases of the chest, making many important contributions to our present-day knowledge of the subject. It was in 1933 that Dr. Graham successfully performed the first pneumonectomy for cancer of the lung on a physician who is still alive today. For the past ten years, he devoted most of his time to an intensive study of the relationship between cigarette smoking and cancer of the lung.

Dr. Graham was chairman of the American Board of Surgery from 1937 to 1941. He was president of the American Surgical Association and the International Congress of Surgery. He was editor of the *Journal of Thoracic Surgery* and the *Yearbook of Surgery*. He also served as chairman of the Board of Regents of the American College of Surgeons. He received many honors during his colorful career. He was awarded the Lister Medal in 1943 when he was made an honorary fellow in the Royal College of Surgeons. The American Medical Association gave him its Distinguished Service Award in 1950. He received the Gold Medal of the American College of Chest Physicians in 1949 and the American Cancer Society's Annual Award in 1951. The French government made him a Knight of the Legion of Honor. He held honorary degrees from many universities in this country and abroad.

One of the most important aspects of his great career was his influence on his colleagues who studied under him. Many surgeons were trained by him who head departments of surgery all over the world. Everyone who had any contact with him was the better for it. He was intensely human, humble and sympathetic, with a good sense of humor. He never compromised with the truth. His interest in civic affairs, in music, and in the under-privileged was intense.

He was one of the all time greats in Medicine.

Alfred Goldman, M.D.
Regent

BOOK REVIEWS

VENOUS RETURN, Gerhard A. Brecher, M.D., Ph.D., Grune & Stratton, Inc., New York and London, 1956

This monograph of 148 pages in eleven chapters should assume the status of a classic because of its thorough coverage of the subject and the obvious authoritative and critical knowledge of this field exhibited by the author. It is written with great simplicity and clarity and abundantly illustrated with diagrams and records of animal and human studies. The summaries and "clinical implications" are concise and pointed. The material has been carefully edited, and competently indexed and the bibliography includes both the important historical and current references.

The author has succeeded in expressing the need for consideration of venous blood flow, especially as affecting cardiac output, as related to but not necessarily dependent on the magnitude of local venous pressures. The relative importance in establishing gradients of "forward" pressure to the negative pressure induced by respiration and cardiac systole and diastole is clearly delineated. The influence is presented of neuromotor tone, partial and complete collapse of veins, hydrostatic pressure and blood depots in maintenance of adequate venous return.

It would seem desirable to the clinician to have a somewhat more extensive discussion of the problems of venous return in right ventricular failure, shock, constrictive pericarditis, and depletion of the venous reservoirs as by venesection, and its influence on precipitation of pulmonary edema.

This monograph, with much direction toward the basic physiologist, furnishes essential fundamental and practical information for the internist and surgeon interested in cardio-vascular disease and should be helpful to the anesthetist.

John J. Sampson, M.D.

PULMONARY EMPHYSEMA, edited by Alvan L. Barach, M.D., and Hylan A. Bickerman, M.D., Williams & Wilkins Company, Baltimore, 1956.

Drs. Barach and Bickerman, in collaboration with 16 additional well-qualified contributors, have combined their efforts to discuss the subject of pulmonary emphysema from all aspects, physiological, pathological, and therapeutic. There is some repetition and some differences of opinion among the contributors and the literature in certain aspects of emphysema and its management, but this, however, only emphasizes the importance and complexity of the subject.

The management of emphysema as it is carried out by the editors in their clinic is presented at the onset. Administration of low concentrations of oxygen with graded increases has avoided some of the objections to oxygen therapy and is physiologically sound. Some difference of opinion exists concerning the role of pressure breathing in the management of emphysema. This reviewer feels that a proper selection of patients is important and agrees with the editors that the routine use of these forms of treatment should be condemned in patients with emphysema. The importance of preserving and restoring cardio-respiratory function is emphasized throughout the volume.

The book is conveniently subdivided and illustrated in such a fashion that any aspect of emphysema can be readily reviewed with a good bibliography provided. Much of the material presented is fundamental and applies not only to pulmonary emphysema, but to all chronic degenerative conditions affecting the lungs and consequently the contributors digress to discuss other pulmonary diseases as well.

The book reflects the experiences of the authors in their understanding and in their basic contributions to the subject. It is highly recommended to internists, thoracic surgeons, physiologists, and anyone treating emphysema patients. It should provide an excellent reference for students, interns, and residents. Of special merit are the sections on therapy, respiratory acidosis, respiratory physiology and pulmonary function tests.

Joseph F. Tomashefski, M.D.

THE INDEX

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Joseph F. Tomashefski, M.D.

MEDICAL SERVICE BUREAU

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Director, diagnostic and research tuberculosis laboratory, wanted in central Florida. Physician or non-physician microbiologist or clinical chemist. Part-time employment considered. Write: Dr. Albert V. Hardy, Director, Bureau of Laboratories, State Board of Health, PO Box 210, Jacksonville, Florida.

CALENDAR OF EVENTS

NATIONAL AND INTERNATIONAL MEETINGS

Interim Session and Semi-Annual Meeting, Board of Regents,
American College of Chest Physicians
Philadelphia, December 2, 1957

Fifth International Congress on Diseases of the Chest
Council on International Affairs
American College of Chest Physicians
Tokyo, Japan, September 7-11, 1958

POSTGRADUATE COURSES

12th Annual Postgraduate Course on Diseases of the Chest
Hotel Knickerbocker, Chicago, October 21-25

10th Annual Postgraduate Course on Diseases of the Chest
Park-Sheraton Hotel, New York City, November 11-15

3rd Annual Postgraduate Course on Diseases of the Chest
Ambassador Hotel, Los Angeles, December 9-13

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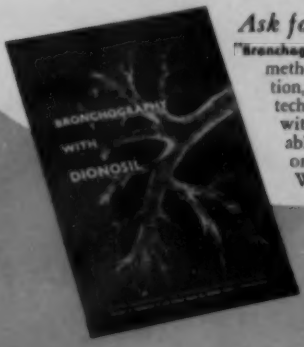

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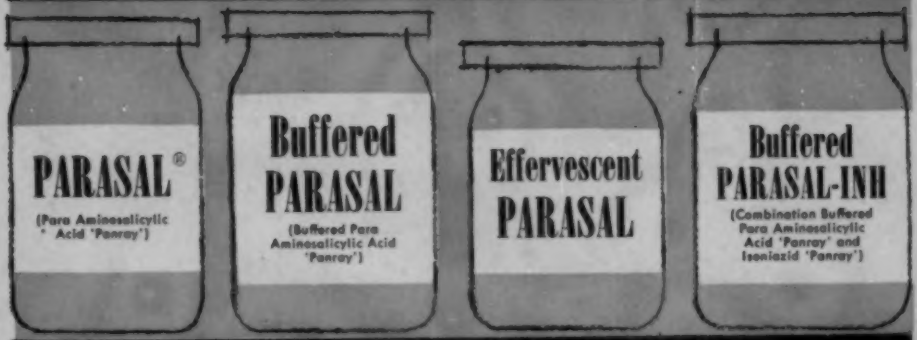

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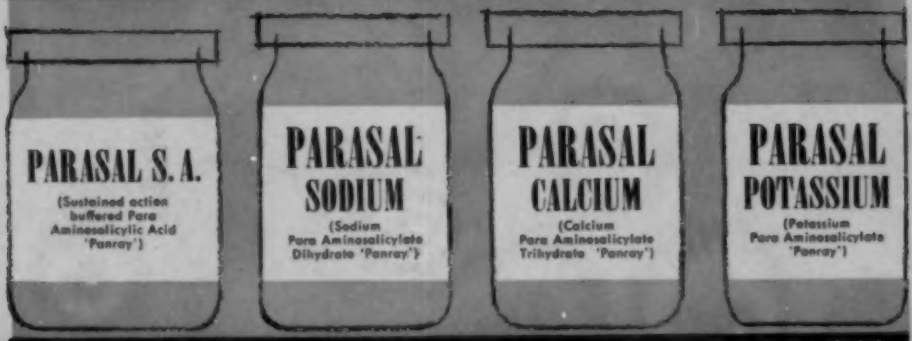

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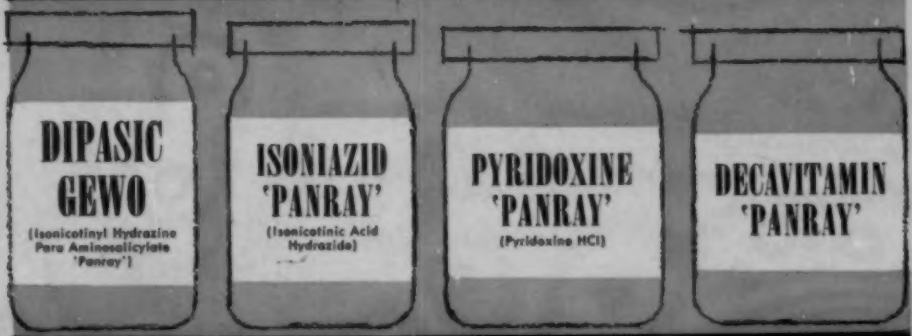

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